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I.

THE OPTIC NERVE AND THE ACCESSORY CAVITIES OF THE NOSE.

CONTRIBUTION TO THE STUDY OF CANALICULAR NEURITIS AND
ATROPHY OF THE OPTIC NERVES OF NASAL ORIGIN.*

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PREFACE.

For ten years I have been investigating the more delicate construction of the accessory cavities, and the relation of the optic nerve to them. The results obtained furnish the anatomic foundation for the theory of sight disturbances and blindness caused by diseases of the accessory cavities, particularly of the posterior ethmoid cells and the sphenoid sinus. An exact pathologico-anatomic foundation for this theory is still to be created. The observations with which we are familiar I have incorporated in the chapter on clinical observations.

*This paper is translated with the authority of Professor Onodi given to the editor of the Annals of Otolaryngology and Rhinology.

The many discrepancies in our knowledge of the subject are mentioned, but the complete establishment of this theory is to be expected from exact clinical observations and pathologico-anatomic research. I offer my colleagues the work of many years in the hope that it will lead to further mutual research in rhinologic and ophthalmologic fields, and to the successful solution of this important question.

My preparations are photographed in natural size, ten of which I used for demonstration before the 33rd Congress of the Ophthalmological Society, at Heidelberg in 1906.

Of the 33 illustrations 8 preparations are used which appear in my atlas on "The Accessory Cavities of the Nose."

TOPOGRAPHIC ANATOMY.

The intimate relation of the optic nerve to the accessory cavities of the nose, and the more delicate topographico-anatomic relations have not, until recently, been exhaustively treated. Generally speaking, the text-books on anatomy pointed simply to the intimate relation of the optic nerve to the sphenoid sinus, which is mentioned, of course, in the description of the course of the optic nerve, and the base of the skull cavity. Berger and Tyrmann¹, in their monograph, speak of the relation of the optic nerve to the sphenoid sinus, and show three instances of the different thicknesses of the sphenoid sinus wall in the region of the foramen opticum. They also mention two skulls, in which the ethmoid labyrinth extends so far back, that the front wall of the sphenoid sinus lies behind the optic nerve canal, so that the latter does not touch the sphenoid sinus. Zuckerkandl² and Douglas³ have described the sphenoid cell in the region of the lesser sphenoid wing. These were the forerunners of my years of introductory and preliminary research. In my publications I have shown the frequently intimate relation of the optic nerve to the last ethmoid cell, and proved in this way that it is about time to drop the almost mechanical assumption of the relation of the optic nerve to the sphenoid sinus. My completed studies have resulted in the establishment and confirmation of 38 different morphologic relations, bearing on the relation of the optic nerve to the sphenoid sinus, and the last ethmoid cell. When we take into consideration the embryonic relation of the canalis and sulcus opticus, and give our attention to the morphologic relations

which the different parts of the sphenoid, such as the presphenoid, the orbitosphenoid, and the basisphenoid show in the fetal and post-fetal periods to the development of the sphenoid sinus, the appearance of the sphenoid turbinate, the development of the ethmoid cells, the frontal, and the appearance of centers of ossification (morphologic relations which may show deviations in the fetal and post-fetal period), then we can properly understand most of the morphologic relations which have been found and described, and explain their peculiarities. We do not wish in this connection to examine the morphologic relations of the optic nerves more closely; it is sufficient to throw light upon the topographic relations of the optic nerves, the canalis opticus, and sulcus opticus, and the close relation of this portion to the last ethmoid cell and to the sphenoid sinus.

The 38 different morphologic relations are divided into twelve groups: the individual ones are described with cuts of preparations made in natural size from photographs. The position and course of the optic nerve in the cranium are first presented and illustrated, then the region of the foramen opticum and the canalis opticus, and finally the different relations of the optic nerve, the sulcus opticus, the foramen opticum, and the canalis opticus to the last ethmoid cell, and the sphenoid sinus.

The region of the foramen opticum is illustrated from a bone preparation by figure 1. Above is seen the fissura orbitalis superior with the lesser wing of the sphenoid, ala parva, on the medial side of which is the foramen opticum, which is continuous with the short canalis opticus. The superior portion of the foramina optica is limited by an inferior border, the limbus sphenoidalis, which marks the boundary of the jugum sphenoidale lying anterior. Between the foramina optica, the limbus sphenoidalis, and the hypophyseal groove, the sella turcica limiting the tuberculum sellae, the sulcus opticus or sulcus chiasmatis may be seen.

Figure 2 shows the optic nerve entering the foramen opticum, the chiasm, and the optic tract. The flattened strands of this optic tract unite and cross in the chiasm between the limbus sphenoidalis and the tuberculum sellae in the sulcus opticus. The optic nerves emerge from the chiasm in order to pass above the ophthalmic artery in the foramen opticum. The figure shows the internal carotid going to the brain, and the origins of the oculomotorius, trochlearis, trigeminus and abducens.

Figures 2 and 104 in my Atlas⁴ show the base of the brain with the attached tractus opticus in situ. Figure 2 shows in sagittal section the position and relation of the tractus opticus to sphenoid sinus. Fig 104 illustrates a preparation in sagittal section which was injected with a ten per cent. formol solution in order to maintain the mucosa of the individual cells in bladder-like form. The maxillary sinus, the frontal sinus, the ethmoid cells and the sphenoid sinus may be seen in situ, and the base of the brain with the attached tractus opticus and its relations to the ethmoid cells.

Figure 3 illustrates in a formol preparation all the structures which appertain to the orbital cavity. The bony wall has been removed, and the mucosa of the accessory nasal cavities may be seen in bladder-like shape, the inferior surface of the frontal lobe of the brain, the optic nerve and the internal carotid in situ. The position of the optic nerve and its close relation to the sphenoid may be observed under the frontal lobe.

Figure 4 illustrates, in a formol preparation, the position of the chiasm, the course of the left optic nerve, as far as the entrance into the bulb. The left optic nerve touches the left sphenoid sinus, otherwise the nerves have no relation with the accessory cavities in their course. The individual accessory cavities may be seen in bladder-like shape in situ, and on one side the mucosa of the roof of the nose, and, on the other, the opened nasal cavity and the relation of the nasal cavities to the periorbital.

Figure 5 illustrates the accessory cavities in situ, in a formol preparation, sagittal section, with the nasal cavity opened from without, and the relation of the optic nerve to the last ethmoid cell, and to the sphenoid sinus. Before we enter into a closer description of the individual morphologic relations it should be stated that in those cases in which the wall of the canalis opticus and of the sulcus opticus is formed by the last ethmoid cell, its wall may be extremely thin, as thin as tissue paper. In one case the partition was 1 mm. thick, another time 2 mm. In those cases in which the wall of the canalis opticus and of the sulcus opticus is formed by the wall of the sphenoid sinus, it was found with various thicknesses, ranging as follows: 1, 2, 3, 4, 5, 9 and 12 mm. We shall later discuss the importance of this fact, and likewise the bony dehiscence of the canalis opticus and the course of the canal in the sphenoid

sinus and in the last ethmoid cell. The length of the canal may reach 6, 8, 10 or 12 mm. *

GROUP I.

1. To this group belong those cases in which the posterior ethmoid cell and the sphenoid sinus are not in close relation with the canalis opticus or sulcus opticus. Figure 77 of the Atlas, already mentioned, shows a preparation in frontal section. The right sphenoid sinus is 12 mm. long, 10 mm. broad, and 9 mm. in height, and the left 9 by 10 by 20 mm. The ostium sphenoidale on each side is 1 mm. The bony stratum between the optic nerves, canalis opticus and sphenoid sinus has a thickness of 8 to 12 mm.

Figure 6 likewise shows a preparation in frontal section in which the bone substance, 6 mm. in thickness, separates the optic nerve from the sphenoid sinus and ethmoid cell. The optic nerve, the sphenoid sinus and maxillary sinus may be seen on the surface of the section.

Figure 76 in our Atlas shows a frontal section at the posterior border of the nasal septum, in which bone substance, 8 mm. in thickness, separates the sphenoid sinus from the hypophysis, above which both optic tracts with the chiasm may be seen.

GROUP II.

The relation of the last ethmoid cell to the optic nerves on both sides may exhibit three forms.

2. The last ethmoid cells constitute on both sides the medial and inferior wall of the canalis opticus. This relation which is shown in figure 7, in frontal section, is the most instructive, as it shows that the sphenoid sinuses have no relation to the optic nerves, and that they lie under the ethmoid cells where their openings may also be seen. In this preparation the posterior ethmoid cells extend to the foramen opticum, and constitute on both sides the medial and inferior walls of the canalis opticus. The ostium sphenoidale and the reduced anterior wall of the sphenoid sinus may be seen beneath the inferior wall of the last ethmoid cell, near the median line. The thin septum between the sphenoid sinus and the posterior ethmoid cell is 20 mm. wide on the right side, and 15 mm. on the left.

3. The right posterior ethmoid cell constitutes the medial wall of the right canalis opticus, the left posterior ethmoid

cell constitutes the medial and inferior wall of the left canalis opticus, and the wall of the left third of the sulcus opticus.

4. The ethmoid cells are represented by a cavity, this is merged on the left side with the frontal, and these together form a common cavity. The figures 97 and 98 in the Atlas illustrate this preparation in transverse section. It deals with a structural anomaly, the tissue everywhere shows normal conditions, and the openings of the so-called cavities on both sides are evenly and systematically arranged. On the left the frontal merges with a cavity taking the place of one of the ethmoid cells; the length of this common cavity measures 62 mm. On the right the frontal is separated from the cavity representing the ethmoid cell; the cavity is 45 mm. long, the frontal is 23 mm. long. The anterior wall of the left frontal is 7 mm., that of the right one 1.5 mm. thick.

Separated from the anterior wall by 22 mm. lies the foramen ethmoidale anterius, which takes a downward course along the lateral wall of the frontal, continuing in a semicanalis ethmoidalis, 16 mm. in length. The greatest width of the cavity taking the place of the ethmoid cell is 25 mm. On the left, the frontal is 45 mm., on the right 40 mm. wide; on the left it is 65 mm., on the right 40 mm. in height. The frontal opens on both sides with an orifice 9 mm. long and 4 mm. wide above the anterior terminus of the hiatus semilunaris. The processus uncinatus is well developed; the bulla ethmoidalis well arched. The sphenoid sinus opens on the posterior part of the hiatus semilunaris. The superior turbinate arises as a projection from the middle turbinate; between them the ethmoid cell, bilaterally symmetrical, opens into the superior meatus through small round orifices. A sphenoid sinus exists on the right side. On the right side the frontal shows a recess, the cavity taking the place of the ethmoid cells, two recesses. The lamina cribrosa is 12 mm. long, 2 mm. wide, and constitutes the center of the roof of the nasal cavity. The cavity representing the right ethmoid cell constitutes the inferior and medial wall of the right canalis opticus, and the wall of the right and middle third of the sulcus opticus; the cavity representing the left ethmoid cell, which merges with the frontal, constitutes the medial wall of the left canalis opticus, and the wall of the left third of the sulcus opticus.

GROUP III.

The relations of the posterior ethmoid cell on one side to the optic nerve show seven forms, five of which concern the same side, two the opposite side.

5. On the same side the last ethmoid cell constitutes the medial, inferior and lateral wall of the *canalis opticus*, also the wall of the right third of the *sulcus opticus*. In the Atlas, figure 17 shows a bone preparation in the sagittal section, illustrating this relation. On the right side, one sees that the otherwise large sphenoid sinus bears no relation to the foramen opticum, *canalis opticus* or *sulcus opticus*. The sphenoid sinus is 22 mm. long, 18 mm. high, the posterior ethmoid cell is 30 mm. long; beginning at the median line, it is 18 mm. wide. The latter extends under the *sulcus* and the foramen opticum into the small sphenoid lobe, and constitutes the whole wall of the *sulcus opticus*, also the medial, inferior and lateral wall of the *canalis opticus*.

6. On the same side the posterior ethmoid cell constitutes the medial and inferior wall of the left *canalis opticus*, and the whole wall of the *sulcus opticus*.

In the Atlas, figure 16 shows a bone preparation in the sagittal section, illustrating this relation. On the left side one sees that the otherwise large sphenoid sinus stands in no relation to the foramen opticum, *canalis opticus*, or *sulcus opticus*. The sphenoid sinus is 25 mm. long, 15 mm. high, and 15 mm. wide, from the median line; the last ethmoid cell is 11 mm. long, 14 mm. wide from the median line, and 13 mm. in height. The posterior ethmoid cell constitutes the entire wall of the *sulcus opticus*, also the medial and inferior wall of the *canalis opticus*.

7. On the same side the last ethmoid cell constitutes the inferior and medial wall of the *canalis opticus*. Figure 13 of the Atlas shows a bone preparation with the sagittal section illustrating this relation. On the left side is seen the last ethmoid cell, 34 mm. long and 16 mm. high, and its relation to the optic nerve; the ethmoid cell extends back 6 mm., until close under the *canalis opticus*. The accessory and nose cavities are opened from the outside.

8. The last ethmoid cell constitutes on the same side the upper, medial and inferior wall of the *canalis opticus*. Figures 8 and 9 illustrate this relation. Figure 8 shows by a sagittal section how the last right ethmoid cell surrounds the *canalis opticus*

medially, above and below. Figure 9 illustrates a preparation, a frontal section opening the last ethmoid cell in front; it was also opened through the nasal cavity. On the right side one sees the entrance of the optic nerve into the foramen opticum, next to the carotis interna, and the canalis opticus, 12 mm. long, taking its course into the last ethmoid cell, which surrounds it above, below and medially. On the same side the last ethmoid cell constitutes the superior, inferior, medial and lateral wall of the canalis opticus.

9. The last ethmoid cell forms on the same side the upper, lower, medial and lateral walls of the canalis opticus. Figure 10 illustrates this relation in a sagittal section, on the right side is seen the canalis opticus in the last ethmoid cell, the entrance and departure of the optic nerve into and out of the canalis opticus. The length of the canalis opticus is 10 mm.; it disappears into a posterior ethmoid cell 15 mm. long and 18 mm. in height.

10. The last ethmoid cell constitutes, on the same side, the medial wall of the canalis opticus. Figure 11 illustrates this relation in a sagittal section. On the left side is seen the entrance of the optic nerve into the canalis opticus; the thin partition wall, which surrounds the optic nerve medially, has been removed in the ethmoid cell; the optic nerve, for 6 mm. in length, touches the ethmoid cell.

11. The posterior ethmoid cell constitutes on one side the inferior wall of the canalis opticus. Figures 12 and 13 illustrate this relation. Figure 12 shows by a sagittal section on the left side the course of the optic nerve above the last ethmoid cell; its length is 7 mm.; the thin partition wall has been removed. Figure 13 illustrates by a frontal section the course of the optic nerve above the posterior ethmoid cell. Its length is 4 mm. The thin partition wall has been removed.

12. The right posterior ethmoid cell constitutes on both sides the medial and inferior wall of the canalis opticus, and the entire wall of the sulcus opticus. Figure 14 in the Atlas illustrates this relation, and figure 14 in corresponding sagittal sections. The right posterior ethmoid cell is 17 mm. high, 22 mm. long, and 38 mm. wide. The right sphenoid sinus 14 mm. high, 22 mm. long and 28 mm. wide. The right posterior ethmoid cell is of extraordinary dimensions, it lies above the sphenoid sinuses and bears an intimate relation to the chiasma and both nervi optici, because the entire sulcus opticus and the inferior wall

of the canalis opticus are separated from the posterior ethmoid cell by an exceedingly thin, bony wall. The left sphenoid sinus is 26 mm. long, 20 mm. wide and 14 mm. high.

13. The left posterior ethmoid cell constitutes the medial wall of the right canalis opticus. Figure 53 of the Atlas shows a preparation illustrating this relation in a sagittal section.

The sphenoid sinuses show an asymmetry, owing to the fact that the left sphenoid sinus lies anterior to the right sphenoid sinus. The right sphenoid sinus is 18 mm. long, 15 mm. wide, 16 mm. in height; the left sphenoid sinus is 15 mm. long, 16 mm. wide, 17 mm. in height. The left posterior ethmoid cell is 16 mm. long, 15 mm. broad; it constitutes the medial wall of the right canalis opticus.

GROUP IV.

The sphenoid sinuses on both sides show a close relation to the optic nerves, illustrated by five morphologic relations.

14. The sphenoid sinuses constitute on both sides the medial and inferior wall of the canalis opticus, and the wall of the sulcus opticus. Figure 15 illustrates this relation in a formol preparation. The bony walls of the accessory cavities in the anterior and middle fossae have been removed, and looking from before backwards is seen in situ the mucosa of the frontalis, the ethmoid cells and the sphenoid sinuses in bladder-like form, also the root of the cranial nerves. The sphenoid sinuses show an asymmetric development. The left sphenoid sinus extends backwards towards the right and the right sphenoid sinus in front extends towards the left. The sphenoid sinuses touch medially and underneath the corresponding optic nerves.

15. The sphenoid sinuses constitute on both sides the medial wall of the canalis opticus, and the wall of the corresponding third of the sulcus opticus. Figure 75 of the Atlas illustrates this relation by a frontal section. Anterior to the sphenoid sinus the optic nerves are seen entering the canalis opticus. Figure 16 illustrates the same relation by a frontal section. The extremely thin partition wall is seen. The sphenoid sinus on both sides is 17 mm. wide, 22 mm. high, on the right 22 mm. long and on the left 25 mm. long. Figure 17 illustrates by a frontal section the optic nerves on both sides bounded by the lateral wall of the sphenoid sinus, and on the right bordered by the inferior wall of the posterior ethmoid cell.

16. The sphenoid sinuses on both sides constitute the inferior wall of the canalis opticus, and the wall of the sulcus opticus.

Figure 18 illustrates this relation by a frontal section, with this difference, the left sphenoid sinus shows a greater development and it constitutes the wall of the left and middle third of the sulcus opticus.

17. The sphenoid sinuses constitute on both sides the wall of the sulcus opticus.

18. The right sphenoid sinus constitutes the inferior and medial wall of the right canalis opticus, and the wall of the sulcus opticus; the left sphenoid sinus constitutes the inferior wall of the left canalis opticus.

GROUP V.

The relation of the sphenoid sinuses to the optic nerve show seven forms, of which four are on the same side, two on the opposite, and one in the center.

19. The sphenoid sinus on the left side constitutes the inferior wall of the left canalis opticus, and the wall of the entire sulcus opticus.

20. The sphenoid sinus of the left side constitutes the inferior wall of the left canalis, and the wall of the left third of the sulcus opticus. This relation may also occur on the right side.

21. The right sphenoid sinus constitutes the inferior wall of the canalis opticus.

Figure 19 illustrates this relation, a formol preparation of which the bony walls of the right orbit have been removed; the individual accessory cavities and the optic nerve in situ are seen and the above mentioned relation of the optic nerve to the sphenoid sinus is also visible.

Fig. 20, a formol preparation, shows the intimate relation of the left optic nerve to the left sphenoid sinus, which, 12 mm. long, constitutes by an extremely thin bony stratum, the inferior wall of the canalis opticus. The chiasma and the right optic nerve are separated from the sphenoid sinus by sponge-like bones.

22. The right sphenoid sinus constitutes the medial wall of the right canalis opticus. Fig. 21 illustrates this relation by a sagittal section. The thin joint partition wall between the canalis opticus and the sphenoid sinus has been removed, and

the optic nerve is seen taking its course, 5 mm. in length, along the lateral wall of the sphenoid sinus.

23. The sphenoid sinus of the right side constitutes the medial and inferior wall of both canales optici, and the entire wall of the sulcus opticus. Figures 22 and 23 illustrate this relation.

Figure 22, a formol preparation, shows from above the close relation of the right sphenoid sinus to both optic nerves, while Fig. 23 illustrates by a corresponding section the extraordinarily great asymmetry of the sphenoid sinus; the left sphenoid is small, and pushed completely aside.

24. Figure 24 is a preparation illustrating a frontal section; the right sphenoid sinus shows a close relation only to the left optic nerve, which is bordered medially by the extremely thin wall of the right sphenoid sinus.

25. The sphenoid sinus of the left side constitutes the wall of the middle third of the sulcus opticus. Figure 25 illustrates this morphologic relation by a frontal section, showing the left sphenoid sinus in the median line above the nasal septum.

GROUP VI.

The sphenoid sinus and the posterior ethmoid cell on the same side show a close relation to the optic nerves in two forms. One form affects the optic nerve of the same side, the other both optic nerves.

26. The last posterior ethmoid cell of the left side constitutes the medial wall, and the left sphenoid sinus, the inferior wall of the left canalis opticus.

Figure 26 illustrates this relation by a formol preparation. The nasal cavity has been opened from without, the last posterior ethmoid cell, marked by a cross, is seen; also its relation and that of the sphenoid sinus to the optic nerve of the same side.

27. The left posterior ethmoid cell forms the medial wall of the left canalis opticus, and the left sphenoid sinus the inferior wall of the right canalis opticus, also the wall of the right and middle third of the sulcus opticus.

Figure 27 illustrates this relation by a formol preparation of which the inner wall of the orbital cavity has been removed; the mucosa of the accessory cavities is visible. The preparation also illustrates the relation of the left posterior ethmoid cell (marked by a cross), to the left optic nerve, and

of the left sphenoid sinus to the right optic nerve. The same relation may exist on the right, also the left sphenoid sinus may be absent.

GROUP VII.

The last ethmoid cells and the sphenoid sinuses on both sides show relations to the optic nerves in one form.

28. The last posterior ethmoid cells constitute on both sides the medial wall of the canalis opticus, and the sphenoid sinuses on both sides the walls of the sulcus opticus.

GROUP VIII.

The last posterior ethmoid cells on both sides, and the sphenoid sinus on one side, show a relation to the optic nerves in two forms.

29. The right posterior ethmoid cell constitutes the inferior and medial wall of the right canalis opticus, and the wall of the right third of the sulcus opticus; the left posterior ethmoid cell constitutes the medial and inferior wall of the left canalis opticus, also the wall of the left third of the sulcus opticus; the right sphenoid sinus constitutes the middle third of the sulcus opticus. This relation is illustrated by figure 67 in the Atlas; it is a frontal section, where in the median line the right sphenoid sinus is visible above the nasal septum. This spur of the sphenoid sinus extends from the ostium sphenoidale 15 mm. to the front, and stands in relation to the middle third of the sulcus opticus and the chiasma.

30. The left sphenoid sinus constitutes the wall of the left third of the sulcus opticus; the left posterior ethmoid cell constitutes the medial and inferior wall of the canalis opticus; the right posterior ethmoid cell constitutes the medial, anterior, lateral and inferior wall of the right canalis opticus, and the wall of the right third of the sulcus opticus.

GROUP IX.

The sphenoid sinuses on both sides and the posterior ethmoid cell on one side, in their bearing on the optic nerves show one form.

31. The left sphenoid sinus constitutes the medial wall of the left canalis opticus; the right sphenoid sinus constitutes the medial wall of the right canalis opticus; the right posterior ethmoid cell constitutes the anterior wall of the right canalis opticus.

GROUP X.

The sphenoid sinus on one side, and the last posterior ethmoid cell on one side, in their relation to the optic nerves show four morphologic relations.

32. The left sphenoid constitutes the medial and inferior wall of the left canalis opticus and the wall of the entire sulcus opticus, also a small part of the inferior and medial wall of the right canalis opticus, whereas the right posterior cell forms the greater part.

33. The left posterior ethmoid cell constitutes the medial, anterior, lateral and inferior wall of the left canalis opticus; the right sphenoid sinus constitutes the medial, anterior, lateral and inferior wall of the right canalis opticus.

Figures 28 and 72 of the Atlas illustrate this relation by corresponding frontal sections. Both instructively show the relation of the optic nerve and the canalis opticus on the one side to the sphenoid sinus, and on the other side to the posterior ethmoid cell; furthermore, the course of the canalis opticus in these cavities, 12 mm. long. The posterior ethmoid cell is 28 mm. long, 30 mm. wide, 26 mm. high; the sphenoid sinuses show an extraordinary asymmetry, the sphenoid sinus on the left side is 30 mm. wide, 22 mm. high, 41 mm. long, on the right side 20 mm. wide, 22 mm. high, and 39 mm. long. The anterior end of the left sphenoid sinus lies asymmetrically in the median line between the lamellae of the septum of the nose.

34. The left sphenoid sinus forms the medial and inferior wall of the left canalis opticus, and the wall of the left and middle third of the sulcus opticus; the right posterior ethmoid cell constitutes the medial and in part the anterior wall of the right canalis opticus.

35. The left posterior ethmoid cell constitutes the medial and inferior wall of the left canalis opticus, the wall of the entire sulcus opticus, and the inferior wall of the right canalis opticus, the right posterior ethmoid cell constitutes the medial wall of the right canalis opticus.

GROUP XI.

The frontal sinus shows two morphologic relations to the optic nerve on one side.

36. The frontal on the same side constitutes the superior wall of the canalis opticus.

37. In the second group, that case was mentioned and described in which the cavity representing the left ethmoid cell merges with the left frontal, and the common cavity constitutes the medial wall of the left canalis opticus and the wall of the left third of the sulcus opticus.

Figures 97 and 98 of the Atlas illustrate this relation by a cross-section.

GROUP XII.

The relation of the turbinate cell of the superior turbinate on one side to the optic nerve shows one form.

38. The turbinate cell, so-called bone-cyst, of the left superior turbinate constitutes the inferior wall of the left canalis opticus and the wall of the left third of the sulcus opticus. This relation is illustrated in frontal section by figure 29. This turbinate cell is 23 mm. long, 19 mm. wide, and 13 mm. in height.

Following the description and illustration of the thirty-eight morphologic relations, which have been arranged in twelve principal groups, we will now discuss certain anatomic facts which are of practical importance and which are to be considered in an etiologic relation. First, the bony walls of the canalis opticus, and of the sulcus opticus; second, the dehiscence of the wall of the accessory cavities; third, the semicanalis ethmoidalis; fourth, the common partition walls of the individual accessory cavities, and fifth, the turbinate cell, so-called bone-cyst, play an important part in diseases of the accessory cavities which cause impaired sight and blindness. Each of these morphologic relations will be separately discussed.

I. THE BONY WALLS OF THE CANALIS OPTICUS AND OF THE SULCUS OPTICUS.

The strength and thickness of those bony walls of the accessory cavities which border the optic nerve, may, in the region of the optic nerve, aid, make difficult or hinder the destruction, impaired circulation, pressure, fracture, and the transmission of the disease. Berger and Tyrmann⁴ first examined the differences in the partition wall between the optic nerve canal and sphenoid sinus. They made frontal sections in the plane of the foramina optica, and showed by three illustrations the varying strength and thickness of the bony wall. The first figure illustrates a case in which the partition walls

between the optic nerve canal and the sphenoid sinus on both sides are unusually thin. The second illustration shows a preparation of which the spongy partition wall between the optic nerve canal and the sphenoid sinus on both sides is 4-6 mm. thick. The third figure illustrates a case in which the partition wall on one side is thin as paper, on the other it has a thickness of 7 mm. Our observations have shown that the wall between the last ethmoid cell and the canalis opticus and the sulcus opticus is nearly always as thin as tissue paper; in one case it was 1 mm., in another 2 mm. thick. This extremely thin wall between the last ethmoid cell and the optic nerve, figure 30 illustrates by a sagittal section. We have often found a thicker section between the sphenoid sinus and the canalis opticus and the sulcus opticus, the thickness varying between 1, 2, 3, 4, 5, 6, 9 and 12 mm. Figures 31-77 of the Atlas illustrate the extremest differences. Figure 31 illustrates by a frontal section the thin septum between the sphenoid sinus and the optic nerve. Figure 77 illustrates by a frontal section the thickest wall we have observed between the optic nerve, canalis opticus, sulcus opticus and the sphenoid sinus. Figure 32 shows in the region of the optic nerves, a bony wall 6 mm. in thickness, which separates the optic nerve from the sphenoid sinus, and Figure 76 of the Atlas illustrates a frontal section, where bone substance 8 mm. in thickness, separates the chiasma from the sphenoid sinus. In those cases in which the canalis opticus takes its course in varying lengths into the sphenoid sinus or the last ethmoid cell, it constitutes a thin bony wall. Figure 9 illustrates a preparation in which the canalis opticus makes its way, 12 mm. in length, into the last ethmoid cell, which borders it above, below and medially. Figure 28, in the text, and figure 72 of the Atlas, show the course of the optic nerve into the cavities and the thin wall of the canalis opticus. Figure 28, a frontal section, shows on the right side the optic nerve and the canalis opticus in the sphenoid sinus; and on the left side the optic nerve and the canalis opticus in the last ethmoid cell. The canalis opticus, takes its course into the cavities for 12 mm. on the right bordered by the sphenoid sinus, on the left, above, below, medially and laterally by the last ethmoid cell.

Figure 10 illustrates by a sagittal section the optic nerve and the canalis opticus in the last ethmoid cell. Completely bor-

dered by the last ethmoid cell, as is the optic nerve, the canalis opticus, 10 mm. in length, takes its course into the same.

II. THE DEHISCENCE OF THE WALLS OF THE ACCESSORY CAVITIES.

The practical significance of the bony dehiscence is proven by the fact that they may aid or cause orbital and intracranial disturbances as well as sight derangements. The diseased mucosa of the accessory cavities may come in direct contact with the periorbita, the dura mater and the optic nerve sheath and in this way the disease is easily transmitted to the parts mentioned. The dehiscences of the vessel grooves may also lead to a diseased condition of the same. The dehiscences may be the consequences of pathologic changes, senile atrophy, trauma; we may also have to do with artefacts and formative anomalies. We have already discussed these conditions in an article.⁵ We will now confine ourselves to anomalies of construction and congenital dehiscences. Such dehiscences have not come under our notice. Zuckerkandl⁶ mentions four, Merlin⁷ two cases. In the cases observed by Zuckerkandl, the dehiscences of the sphenoid sinus were connected by congenital dehiscences of the lamina papyracea of the ethmoid sinus; all four cases showed dehiscences of the orbital wall of the sphenoid sinus, one case having at the same time a dehiscence of the tuber maxillae. In two cases Merlin observed this dehiscence on the inferior orbital wall. Taking the dehiscences of the frontal into consideration, we have observed in part the extreme attenuation of the superior wall of the frontal between the lamellae of the superior orbital wall, the dehiscences being in part lineal, part dot-like in appearances. Congenital dehiscences and vessel openings may occur on the anterior wall of the frontal. Zuckerkandl and Helly,⁸ each had a case under his observation in which a furrow taking its course from the medial part of the arcus superciliaris upwards and in part broken, communicated with the frontal. Under our observation a case in vivo showed this congenital dehiscence, a polyp coming through, or penetrating it from the frontal. Helly noticed vessel openings which connected the frontal with the anterior surface of the frontal bone. We have also observed such vessel orifices in the shape of larger or small holes, in the region of the margo supraorbitalis, and the arcus

supraorbitalis. To the congenital dehiscences of the frontal belong those rare cases in which dehiscences of the lamina papyracea of the ethmoid bone open the orbital wall of the frontal at the same time. Zuckerkandl observed this dehiscence in three cases, twice on the left, once on the right side. Merlin observed it once on the left side. We saw three cases of this dehiscence, twice on the right, once on the left side. Concerning the dehiscences of the septal walls of the frontal, according to the observations of Denouvilliers and Gosselin,⁹ Winkler¹⁰ and Killian,¹¹ congenital dehiscences in the form of holes or openings of various sizes may occur on the septum interfrontale. We saw one case in which there was an opening 1.5 mm. in circumference, with smooth edges on the septum interfrontale. Concerning the dehiscence of the lamina papyracea of the ethmoid bone, Hyrtl,¹² already mentions it. Zuckerkandl has observed fourteen cases in which the congenital dehiscence appeared nine times on the left side. Merlin found congenital dehiscences three times in the lamina papyracea of the ethmoid bone, twice on the left, once on the right side. In one case five dehiscences of the lamina papyracea connected the ethmoid cells with the orbital, frontal and sphenoid sinus. Eighteen cases of congenital dehiscences of the lamina papyracea of the ethmoid bone have come under our notice, thirteen being on the left and five on the right side.

The sagittal diameter measured between 4 and 28 mm., the vertical between 4 and 8 mm. In consequence of the dehiscence the ethmoid cells communicated with the orbital cavity; in three cases the frontal, which communicated with the ethmoid cells and the orbital cavity, was also opened.

Figure 23 of the Atlas illustrates a preparation with a dehiscence of the lamina papyracea. Concerning dehiscences of physiologic origin of the sphenoid sinus wall, Zuckerkandl observed on the lateral wall small openings, dehiscences, which connected the sphenoid sinuses with the middle skull cavity. In one case Spee¹³ noticed a defect of the sulcus caroticus. On a number of skulls he noticed vessel openings, sometimes symmetrically arranged on both sides, directly under the lateral root of the lesser sphenoid wing. In several instances vessel grooves lead to these vessel openings, small, large and long dehiscences occurring. Zuckerkandl and Hajek have observed dehiscences on the septal wall of the sphenoid sinuses. Dehiscences rarely appear on the walls of the canalis opticus:

out of two hundred cases Gallmaerts¹⁴ observed two; Holmes,¹⁵ in fifty cases, also saw two dehiscences. Of the three hundred cases under our own notice but one showed a dehiscence of the canalis opticus.

III. THE SEMICANALIS ETHMOIDALIS.

I have described a half or semi-canal, the semicanalis ethmoidalis, which in varying lengths takes its course from the foramen ethmoidale anterius along the wall of the frontal, or the orbital cells to the anterior skull cavity. Through this semi-canal the ethmoidal veins take their course free, and protected by mucosa into the above-mentioned cavities. At the respective ends of the semi-canal the mucosa touches the orbital periosteum and the dura mater. In consequence of this connection transmission of the inflammation may take place, the disease progressing in both directions. Circulatory derangements may also take place, the ethmoidal veins which stand in close relation to the dural venous plexus and the plexus ophthalmicus may be subject to a thrombophlebitis, the same being transmitted to the dural and orbital vein plexuses. Besides the formation of the thrombophlebitis, caused by nervous anastomosis (described by Zuckerkandl and Kuhnt), the ethmoidal vein taking its course into the semicanalis ethmoidalis, may lead to a direct thrombophlebitis, and also to orbital and cerebral complications. Generally speaking, the corresponding marginal grooves of the frontal and the ethmoidal constitute normally a very short canal, the canalis ethmoidalis, which has its beginning in the foramen ethmoidale anterius, ending in the anterior skull cavity with an oblong fissure.

Our examinations in eleven cases showed varying lengths of the canalis ethmoidalis; it varied between 5 and 12 mm. Figure 40 in our Atlas shows by a sagittal section the semicanalis ethmoidalis in an orbital cell. We have designated as orbital cells, those ethmoid cells, which, spread out in the region of the horizontal part of the frontal. One or two orbital cells may occur which open into the middle or upper part of the nasal cavity. The sagittal section of this figure shows the entire course of the semicanalis in a large orbital cell. Inside, it connects the orbital cell with the anterior skull cavity, outside, with the orbital cavity. The foramen ethmoidale anterius extends into the semi-canal, the semicanalis

ethmoidalis, arched-shaped on the posterior and medial wall of the orbital cell, 10 mm. long, and leading to an oblong fissure which opens on the anterior canal cavity. We have observed the semicanalis ethmoidalis three times in the frontal; its length varied from 5 to 8 mm.

In the first orbital cell it appeared nine times, its length was from 7 to 10 mm. In the second orbital cell the semicanalis ethmoidalis appeared four times, from 4 to 10 mm. long. In one case the semicanalis ethmoidalis showed a dehiscence between the orbital cells, thereby causing communication between them. Two cases showed a congenital dehiscence of the lamina papyracea, and the presence of the 8-10 mm. long semicanalis ethmoidalis in the orbital cells. Figure 97 of the Atlas illustrates a case in which the ethmoid cells formed a cavity, which merged into the frontal. The foramen ethmoidale anterius was 23 mm. from the anterior wall of the frontal, and took a downward course along the lateral wall of the frontal as a semicanalis ethmoidalis 16 mm. in length.

IV. THE COMMON SEPTA OF THE INDIVIDUAL ACCESSORY CAVITIES.

In view of our personal observations, we now wish to discuss the thin, in fact extraordinarily thin walls of the septa of the individual accessory cavities; they are of practical importance because of the tendency to the transmission of diseases and to perforations. The maxillary sinus may have a common partition wall with the sphenoid sinus and the ethmoid cells. I have proved that the maxillary sinus and the sphenoid sinus may be so enlarged that they lie next to one another. Figure 90 of the Atlas shows this relation by a cross-section. The maxillary sinus is 42 mm. long, 36 mm. wide, the sphenoid sinus 37 mm. to 47 mm. wide and 35 mm. long. The thin partition wall between the cavities is enlarged to a size of 10 mm., and at the same time constitutes the pars maxillaris of the anterior wall of the sphenoid sinus. We have observed six cases of this common partition wall. Its width, i. e., the pars maxillaris of the anterior wall of the sphenoid sinus, measured 4 mm. twice, 6 mm. once, 8 mm. once, and 9 mm. twice. This thin common septal wall between the maxillary sinus and the sphenoid sinus, figure 49 of the Atlas illustrates by a frontal section. Thin, common septa exist between the maxillary sinus and the ethmoid cells. Figure 63 of the Atlas illustrates by a frontal

section on both sides the common septa between the maxillary sinus and the last ethmoid cell. An extremely thin partition wall between the maxillary sinus and the last ethmoid cell is illustrated by a frontal section—figure 80 of the Atlas. Figure 96 of the Atlas illustrates by a cross section a preparation, in which the last ethmoid cells on both sides are separated from the maxillary sinus by a common septum. The frontal has common partition walls with the ethmoid cells and the sphenoid sinus. The septa between both frontals, the septum interfrontale, may be sagittal in the median line, or in cases of asymmetry, it may have an oblique direction. Figure 27 of the Atlas illustrates by a sagittal section the asymmetry of the frontals; the left frontal covers the right frontal to the extent of 15 mm., and also extends 30 mm. further upwards. Besides the sagittal septum interfrontale a common horizontal partition wall also exists.

It should be noticed that the frontal on both or only on one side may be lacking, and in consequence there could be no question of a septum wall. The common septa between the most anterior ethmoid cell and the frontal, figure 10 of the Atlas illustrates by a sagittal section. Figures 28, 58 of the Atlas show the common septa between the ethmoid cell (spreading out in the region of the frontal), the bulla frontalis, and the frontal. Figure 28 illustrates a sagittal section in which the thin septum is easy to see between a bulla frontalis 17 mm. long, 22 mm. high, 21 mm. wide, and the frontal sinus 25 mm. long, 6 mm. high, and 34 mm. wide. Figure 58 illustrates by a frontal section the common septum on both sides between the bulla frontalis and the frontal. On the right the frontal is 20 mm. long, 30 mm. wide, 11 mm. high; on the left it is 14 mm. long, 30 mm. wide and 11 mm. high; on the right the bulla frontalis is 20 mm. long, 13 mm. wide, 20 mm. high; on the left it is 20 mm. long, 13 mm. wide, 20 mm. high. The common septum between the orbital cells and the frontal is illustrated by the following sagittal sections. Figure 41 of the Atlas shows the thin arched partition wall between the frontal, which is 23 mm. high, 30 mm. wide, 13 mm. long, and the orbital cell, 27 mm. long, 19 mm. high and 29 mm. wide. Figure 36 of the Atlas shows in a sagittal section the existing communication between the frontal and the orbital cell. Figure 30 of the Atlas illustrates in a sagittal section the extraordinarily thin common septum be-

tween the frontal, 19 mm. long, 35 mm. high, 32 mm. wide, and the orbital cell, which is 25 mm. long, 28 mm. wide and 13 mm. high. Figure 26 of the Atlas illustrates a preparation in which the frontal is completely separated from the last ethmoid cell by a common septum. Figure 7 of the Atlas shows the absence of the septum and the merging together of the frontal with the ethmoid cell space.

Figures 31, 37, 52 and 38 in the Atlas show in sagittal section the common septum between the frontal and both orbital cells, also the communication of the individual cavities. Figure 31 shows a thin partition wall between the frontal and first orbital cell, also a thin partition wall between the first and second orbital cells. Figure 37 shows a perfect partition wall between the first and second orbital cells, and communication between the frontal and first orbital cell. Figure 52 shows a complete septum between the frontal and the first orbital cell, and a communication between both orbital cells. Figure 38 shows the communication between the frontal sinus and both orbital cells. The frontal may extend far backwards; it may even reach the foramen opticum, and this expansion may be separated from the posterior ethmoid cell, as well as from the sphenoid sinus by thin common septa. Figure 65 of my Atlas illustrates in frontal section a preparation, in which the frontal, extending to the rear between the orbital lamellae, forms a common thin wall on the right with the right sphenoid sinus, on the left with the left posterior ethmoid cell. This septum between the frontal and sphenoid sinus, that is the pars frontalis of the anterior sphenoid sinus wall, has a width of 12 mm. Figure 66 of the Atlas illustrates in frontal section a preparation in which the frontal extends posteriorly in the region of the sphenoid sinus and the optic nerve; a thin septum is visible between the frontal and the posterior ethmoid cell.

We have already discussed the common partition walls between the ethmoid cells, frontal and maxillary sinus; the cross-sections in figures 52 and 67 of the Atlas show the septa between the individual ethmoid cells; it is nearly always thin. The common partition walls found between the posterior ethmoid cell and the sphenoid sinus are still to be discussed. The posterior ethmoid cell may rise shelf-like above the sphenoid sinus, or it may be in a position anterior to the sphenoid sinus; the common thin wall between both cavities may be in a vertical

or horizontal position, or in both directions in an oblique position. According to our examinations the width of this common septum, the pars ethmoidalis of the anterior sphenoid sinus wall, may vary from 1 to 18 mm. Figure 67 shows in a cross-section the common partition wall between the posterior ethmoid cell and the sphenoid sinus, also the partition wall between both sphenoid sinuses, in a sagittal position; owing to asymmetry of the sphenoid sinuses the partition wall may have various curvatures and positions, and may be even totally lacking in case there is no sphenoid sinus. Figure 68 of the Atlas shows in a sagittal section the shelf-like position of the posterior ethmoid cell above the sphenoid sinus, and the extremely thin common wall between the cavities. Figure 67 of the Atlas illustrates in a frontal section a rare morphologic relation, namely, the common partition walls of the right sphenoid sinus with both posterior ethmoid cells. Figure 28 of the text also shows in a frontal section the common partition wall of the left posterior ethmoid cell with both sphenoid sinuses.

V. THE TURBINATE CELL, SO-CALLED BONE CYST.

The so-called bone cyst is a structural anomaly¹⁸ known as the turbinate cell. Diseased, these turbinate cells may attain various sizes. They appear in the superior and middle turbinate, and, as the turbinate cell of the superior turbinate, according to our observations, may be in close relation to the optic nerve; it will be seen that the practical value of the turbinate cell is due to pathologic changes and growth, especially through pressure.

In cases under our observation the turbinate cell appeared nine times in the middle and twice in the superior turbinate; the turbinate cells twice opened into the middle meatus, at other times in the superior. Its length varied from 8 to 20 mm., the width from 5 to 14 mm., the height from 7 to 16 mm. Figure 18 of the Atlas shows in a sagittal section a turbinate cell in the middle turbinate, 17 mm. long, 20 mm. high, 14 mm. wide, opening into the superior meatus. Figure 11 of the Atlas shows in sagittal section a turbinate cell in the superior turbinate, which opens into the superior meatus. This turbinate cell is 14 mm. long, 14 mm. high and 9 mm. wide. Figure 29 illustrates in frontal section a turbinate cell in the superior turbinate, 23 mm. long, 19 mm. wide and 13 mm. high, which extends back to the region of the optic nerve.

II. CLINICAL OBSERVATIONS.

It is to be regretted that the knowledge of how the diseases of the accessory cavities cause blindness and sight derangements should not yet be firmly established. Excepting individual microscopic and bacteriologic experiments, which prove the continuity of infection, and its passage through the blood vessels, postmortem findings confirm mechanical lesion by which the tumors of the nasopharynx, the sphenoid sinus, the ethmoid sinus and of the ethmoid bone extend into the region of the optic nerve, destroying it or by pressure causing functional derangements and circulatory changes. The postmortem findings show further traumatic lesion of the optic nerve caused by trauma, fractures, fissures in the region of the sphenoid, also pressure of the optic nerve by hyperostosis of the wing of the sphenoid and contraction of the foramen opticum. The postmortem findings confirm suppurations of the accessory cavities, perforations of the cavity walls, basal meningitis, extra- and intradural abscesses, caries, necrosis, thrombosis of the sinus cavernosus, and of the vena ophthalmica. The postmortem findings are faulty, as it is difficult to prove the results of infection, the causal connection, the microscopic and bacteriologic investigations of the diseased accessory cavities, as well as of the optic nerve are wanting, furthermore the more positive assertions of the relation of the optic nerve to the diseased or healthy accessory cavities are lacking, and lastly the investigations of the venae ethmoidales, vena centralis retinae, and the venous radicals of the diseased accessory cavities.

Having assumed that a pathologico-anatomic foundation must be first created, it is clear that there are available only certain stepping-stones, clinical observations, and problematic assumptions, and therefore, at the present time, we can make use only of observations regarding the clinical theory of sight derangements and blindness, canalicular neuritis and atrophy of the optic nerves. Let our clinical observations be preceded by the following remarks. It is known that oculists have established a large number of causes which bring about neuritis and atrophy of the optic nerve. Granting that the causal connection between diseases of the accessory cavities and sight derangements can, and should no longer be doubted, the question as to the accidental appearance of both diseases may still arise. Both of these diseases may surely exist at the same time. Cases have been observed in which an operation

of the accessory cavities did not influence the unfavorable progress of the neuritis, furthermore, after a refusal to operate a suppuration, a neuritis optica healed spontaneously. Other cases have come under our own observation, in which the oculist could only confirm the existence of neuritis or atropia optica, without ability to give the cause, and the rhinologist could find nothing abnormal. Blindness or derangements of sight, due originally to a totally different cause, may still show independent empyemas of the accessory cavities. I wish to emphasize this, because in such cases the accessory cavities can in no way be held accountable, even assuming there is a causal connection. And as it is a fact that the phenomena of disease may closely follow one another without a real or causal connection, it is well to be strictly critical on the matter. This question, in many cases is difficult, or almost impossible to solve. I have touched upon it because in my opinion false interpretations may be easily made.

We will next give our attention to etiologic causes. Under this head come the mechanical and traumatic lesions of the optic nerve, clearly proven by postmortem findings. The tumors of the accessory cavities may cause, in the region of the optic nerve, direct symptoms of pressure. It is not impossible for the tumor to surround the optic nerve, and the same present a normal appearance; as the cases of Bering¹⁹ and Wicherkiewicz²⁰ and the findings of Ponfick show; furthermore, cases of sarcoma of the sphenoid bone observed by Schmidt-Rimpler and Onodi,²¹ showed that both of the optic nerves remained intact. Through postmortem findings, Virchow²² and Manz²³ were able to trace the cause of blindness back to arrested development of the skull in earliest youth, to a premature ossification of the skull, to a hyperostosis, and in consequence thereof an irregular contraction of the foramen opticum, and pressure on the optic nerve.

Blindness in cases of bradycephalus was, among others, observed by Enslin²⁴ and Goldzieher²⁵. In two cases of blindness we also observed anosmia; there was therefore not only contraction of the foramen opticum, but also of the openings of the lamina cribrosa.

Traumatic injury of the optic nerves and direct and indirect fracture of the canalis opticus are well known. Holder²⁶ observed fifty-three cases of fracture of the walls of the canalis opticus. The etiologic significance of these cases was made evident by postmortem findings.

Infection, the transmission of inflammation, bone dehiscences and derangements of circulation are specially to be mentioned as etiologic phenomena of suppuration of the accessory cavities. We have already spoken of the faulty and unsatisfactory postmortem findings; they establish, in general, the infection, the causal connection of the accessory cavities, suppuration with visible bone destruction, thrombophlebitis, meningitis, orbital and cerebral abscesses. The investigations are wanting, of the changes of the septa, the walls of the accessory cavities, and the vessels, particularly those of the ethmoidal veins, and the vena centralis retinae, and of the optic nerve. We have two accurate observations, made by Orman²⁷ and Hajek,²⁸ who have established the cause of infection by way of the blood vessels and tissue continuity.

The case of Orman showed the presence of an empyema of the sphenoid sinuses, an extradural abscess of the sella turcica, and a thrombosis of the sinus cavernosus on both sides. The bone in the region of the sella turcica was a discolored reddish brown; a microscopic examination showed the following:

The presence of numerous diplococci lying between small particles of pus on the mucosa and periosteal plane of the lining of the sphenoid sinuses, also their presence in its substance, infiltrated by hemorrhagic foci.

Sections through the bone (body of the sphenoid) and the membranes showed the presence of diplococci. This finding showed that infection of the meninges may take place by direct continuity. Added to the inflammation and coagulation of the secretions in the sphenoid sinuses, were a periostitis and otitis purulenta, which, penetrating the bone, caused a periostitis on the cerebral plane, and in that way affected the hard and soft membranes of the brain.

The case observed by Hajek showed the presence of an empyema of the ethmoid labyrinth and meningitis. The section showed a diffuse, fibrous, suppurative, acute pachymeningitis interna, and leptomeningitis of the base and convexity of the brain with acute internal hydrocephalus, inflammation of the left ethmoid cells, edema of the mucosa of the maxillary sinuses. The exudate of the brain showed streptococci pyogenes. Hajek expressed himself after the investigations in the following manner:

"We have to deal with the influx of virulent streptococci

into the inflamed mucous membrane tissues, also with an invasion of streptococci into the circulatory system of the blood vessels. In consequence of the familiar vein anastomoses between the mucosa of the ethmoid labyrinth and the dura mater, the meninges were soon infected, but the bone and its marrow substance were not affected."

Westenhoefer's²⁰ earlier statements concerning the nasal lymphogenic origin of cerebrospinal meningitis has not so far been microscopically confirmed; he recently declared that the lymphogenic origin and the cause of infection must be established by microscopic investigations. My cases, which were subjected to postmortem observation, as well as the collected material are being reviewed from a pathologic standpoint by Dr. B. Entz, assistant in the Pathologico-Anatomic Institute in Budapest. These researches are not yet completed; it was therefore impossible to incorporate them in this article.

For further information on the subject we will make use of our observation and postmortem findings, bearing on the complications of the accessory cavities. We will discuss them quickly (divided into groups) before we go further into the subject of etiologic phenomena.

In cases of maxillary sinus empyema, Foucher³¹ notes orbital phlegmon, thrombophlebitis of the plexus pterygoid-eus and of the plexus ophthalmicus. Mair³² notes caries of the ethmoid bone, perforations of the lamina cribrosa and abscess of the orbital lobe.

Panas³³ found necrosis of part of the orbital roof, suppurative osteoperiostitis of the orbit, and abscess of the frontal lobe. The case of Dmochovszky³⁴ showed numerous perforations of the maxillary sinus, rupture into the sphenoid sinus, suppurative meningitis, intradural abscess, abscess of the frontal lobe.

Westermayer's³⁵ showed rupture into the fossa pterygopalatina, caries, perforation of the large sphenoid wing, abscess of the temporal lobe.

In cases of frontal empyemas the following findings are noted:

In the case of Bousquet³⁶, perforations of the posterior and inferior frontal wall with abscess of the frontal; in the case of Koehler,³⁷ perforation of the posterior frontal wall, intradural abscess and meningitis basilaris; in the case of Schind-

ler³⁸, perforation of the posterior frontal wall, frontal abscess and pyemia; in the case of Zirm³⁹, thrombophlebitis of the ocular veins and of the sinus cavernosus, orbital abscess, suppurations and putrefactions of venous thrombi; in the case of Huguenin⁴⁰, encephalitis and meningitis; of Mueller⁴¹, abscess of the left parietal bone, perforation, communication with an extradural abscess, meningitis on the side of the convexity; Paulsen⁴², perforation of the posterior frontal wall of the dura, intradural abscess, meningitis; Huguenin, perforation of the posterior frontal wall, suppurative pachymeningitis and leptomeningitis, intradural abscess, connective tissue adhesion of the membranes with the brain surface; Knapp⁴³, perforation of the posterior frontal wall, periostitis of the orbital plate of the frontal, the dura adhering or growing to the discolored brain, an incipient frontal lobe abscess; Bourot-Lecard⁴⁴, periostitis of the frontal and orbital plates of the frontal, thrombophlebitis of the ocular veins and of the sinus cavernosus, suppurative meningitis; Redtenbacher⁴⁵, perforation of the posterior frontal wall, frontal abscess, intradural abscess; Lennox-Browne⁴⁶, perforation of the posterior frontal wall, extradural abscess, pachymeningitis and leptomeningitis; Hoppe⁴⁷, congenital defect of the posterior frontal wall, meningitis of the convexity and base of the brain; Krecke⁴⁸, extradural abscess, frontal lobe abscess, rupture into the lateral ventricle; Wallenberg⁴⁹, perforation of the posterior and inferior frontal wall, intradural abscess; Silex⁵⁰, perforation of the posterior and inferior frontal wall, abscess of the brain on both sides; Milligan⁵¹, septic thrombosis, meningitis; E. Fraenkel⁵², thrombophlebitis of the sinus longitudinalis superior with pyemia; Macewen-Millar⁵³, thrombophlebitis of the sinus longitudinalis superior in the frontal region, extradural and intradural abscess, pus coating of the brain surface, pyemia; Roth⁵⁴, external frontal abscess, suppurative meningitis, on the right, encephalitis, on the left, abscess; Carver⁵⁵, thrombosis of the sinus longitudinalis superior, meningitis of the convexity; Cholle⁵⁶, meningitis.

The following are findings in cases of ethmoidal empyema:

In the case of Bigbie⁵⁷, perforation of the lamina cribrosa of the ethmoid bone and the frontal, and frontal abscess; Schaffer⁵⁸, perforation of the lamina papyracea of the ethmoid, periostitis orbitalis, perforation of the superior wall of the or-

bital cavity, frontal abscess; Pauncz⁶⁰, and Jacobasch⁶⁰, abscess of the brain; Ogston⁶¹, Warner⁶², Ewald⁶³, meningitis; Trousseau⁶⁴, necrosis of the ethmoid bone and meningitis; Hajek⁶⁵, meningitis.

In cases of empyema of the sphenoid sinus the findings are as follows:

Horner⁶⁶, blindness and exophthalmus on the right, the section showing caries of the base of the sphenoid bone; and the adjoining parts; Panas⁶⁷, blindness, exophthalmus, phlegmona orbitalis in the course of otitis of the sphenoid bone; Rouge⁶⁸, strabismus divergens, loss of the left half of the field of vision, the postmortem findings showing suppurative periostitis and empyema of the left sphenoid sinus; Raymond⁶⁹, blindness, exophthalmus of both sides, the postmortem showing basal meningitis, an exudation of pus compressing the chiasma, thrombosis of the sinus cavernosus, and of the venae ophthalmicae, caries and perforation of the sphenoid plate; Ortman, extradural abscess of the sella turcica, thrombosis of both sinus cavernosi, periostitis, and empyema of the sphenoid sinuses; Gruenwald⁷⁰, basal meningitis, pus in the sphenoid sinuses and the maxillary sinuses; Vince⁷¹, perforation of the basilar apophysis and meningitis; Thioloix⁷², extradural abscess of the sella turcica, meningitis; Flatau⁷³, perforation of the posterior wall, meningitis; Zoeckendoerfer⁷⁴, meningitis; Pekastovsky⁷⁵, necrosis of the sella turcica, meningitis, thrombosis of the sinus longitudinalis; Scholz⁷⁶, periostitis suppurativa, perforation of the superior and lateral sphenoid sinus wall, meningitis, arrosion of the sinus cavernosus, thrombosis of the sinus caroticus and petrosus dexter, intradural abscess of the middle cranial fossa; Schlagenhauer⁷⁷, softening of the body of the sphenoid, meningitis and thrombophlebitis of the sinus longitudinalis; Pauncz⁷⁸, thrombophlebitis purulenta sinus circularis Ridley et sinuum petros. inf. et sup.; Halasz⁷⁹, empyema of the sphenoid sinus and encephalitis; Schroeder⁸⁰, empyema of the sphenoid sinuses, perforation, meningitis, thrombophlebitis of the sinus cavernosus and petrosus.

In the case of combined empyema of the accessory cavities, the following postmortem findings are noted:

Weichselbaum⁸¹, empyema of the right maxillary and frontal cavity, abscess of the upper eyelid, extradural abscess and

frontal lobe abscess; Gruenwald, empyema of the maxillary and sphenoid sinuses, periostitis suppurativa, and softening of both posterior cornua; Pauncz⁸², empyema of the maxillary sinus and the ethmoid cells on the left, rupture from the maxillary sinus into the orbital cavity, meningitis basilaris, abscess of the brain; Hansberg⁸³, frontal empyema on the right side, ethmoid empyema on both sides, caries of the ethmoid, meningitis, frontal lobe abscess on the left side; Duplay⁸⁴, exophthalmus on the right side, total blindness accompanied by basal meningitis, particularly over the sella turcica, suppurative phlebitis in most of the sinuses, several foci of pus in the orbital cellular tissue, pus in the sphenoid sinuses and in the right ethmoid cells; Russel⁸⁵, diminution of sight, ptosis of the upper eyelid, and fixation of the bulb on the left, the postmortem showing intradural abscess of the middle cranial fossa, empyema of the sphenoid sinuses, and of the posterior ethmoid cells, thrombosis of the sinus cavernosus, petrosus and of the left vena ophthalmica; Vossius⁸⁶, empyema of the sphenoid sinus, and of the left ethmoid labyrinth, with caries of the sphenoid bone, thrombophlebitis of the vena ophthalmica superior, the vena angularis and vena facialis anterior sinistra, suppurative pachymeningitis and leptomeningitis; Holmes⁸⁷, empyema of the frontals, the ethmoid cells, the sphenoid sinuses, diplopia, neuritis optica on both sides, abscess of the right frontal lobe; Demarquay⁸⁸, marked drooping of the eye, the eye fixed, not sensitive to light on the right side, the postmortem showing the sinus cavernosus bathed in pus, the sphenoid, ethmoid, and maxillary sinuses filled with pus; Panse⁸⁹, empyema and tubercular granulation in the frontals, ethmoid cells, maxillary and sphenoid sinuses, neuroretinitis on both sides, tuberculosis of the ethmoid bone and anterior portion of the sphenoid, the orbital roofs on both sides carious and covered with a caseous mass, local meningitis, hydrocephalus internus, caseous pneumonia; Leber⁹⁰, nasal tumor, empyema of the sphenoid sinus, blindness, atrophy of the optic nerve, suppurative meningitis and thrombophlebitis of the brain sinus; Finlag⁹¹, empyema of the sphenoid sinuses and ethmoid cells, thrombophlebitis of the sinus cavernosus and circularis.

As the postmortem findings have shown us, the complications stand in close relation to the infection. That infection may

take its course through tissue continuity and the blood vessels has been proven, that it may make its way through the lymph vessels, along the lymph spaces of the olfactory nerves through the openings of the lamina cribrosa, is possible, but has not been, up to date, microscopically and bacteriologically confirmed. The transmission of the inflammation, the diseased processes of the accessory cavities, the pathologic changes of the contents of the orbit, the canalis opticus, the optic nerve, the intracranial and cerebral complications stand in close relation to the venous system and to other important etiologic conditions. To the circulatory disturbances belong hyperemia, edema of the tissues, the optic nerve and its sheath, hemorrhages, embolisms; thrombophlebitis and thrombosis. We combine as one those connections between the venous radicals and the venous plexus, which are of practical value and which have made clear to us the origin of circulatory disturbances, as well as of thrombophlebitis and thrombosis. Zuckerkandl's investigations have shown that the veins of the nasal mucosa bear a relation to the facial veins, the anterior ethmoidal veins to the dura and pia mater; the venous radicals of the nasal mucosa to those of the pharynx and palate and to the venous plexus of the fossa pterygopalatina. We are familiar with the connection existing between the venous plexus of the nasolachrymal canal and lachrymal sac and the vena facialis anterior, the vena ophthalmica and the vena infra-orbitalis.

The vena lacrimo-facialis, described by Zuckerkandl, stands in relation to a larger branch, which has its origin in the anterior ethmoid cells, and which bores its way through the lachrymal bone. The venous plexus of the maxillary sinus bears a relation to a branch which bores its way through the sphenoid sinus wall, and with the vena ophthalmica facialis. According to Kuhnt⁹², the venae perforantes of the frontal bring it into a relation with the dural venous plexus. Kuhnt⁹³ and Gurwitsch⁹³ found connections between the veins of the frontal, the vena ophthalmica superior and vena supra-orbitalis. The vena ethmoidalis anterior and posterior empty into the vena ophthalmica superior. The vena ethmoidalis anterior may be joined with the vena angularis and the vena supraorbitalis. According to Onodi, the vena ethmoidalis anterior may lie bare in the semicanalis ethmoidalis in a course

of varying length, and in the frontal and first and second orbital cells. The *venae ophthalmicae superiores* are connected with the *venae ethmoidales*, the *vena centralis retinae*, and also the *vena ophthalmica inferior*. The veins of the eye are connected with the facial veins, the veins of the nasal cavity, the plexus pterygoideus, as well as the sinus cavernosus. The *vena centralis retinae* may be directly joined to the sinus cavernosus. The veins of the sphenoid sinuses may stand in direct relation to the plexus ophthalmicus and the sinus cavernosus. According to Kraus⁹⁴ it is possible that the *vena ophthalmica inferior* or a branch may make its way out into the fossa pterygopalatina, furthermore, that the central vein of the opticus may take its way through the inferior fissure. Owing to numerous anastomoses the circulatory disturbances are easily explained. Hyperemia and symptoms of congestion are also explained by reason of these vein connections. The transmission of inflammation to the orbital tissue and to the sheath of the optic nerve may cause lesion and pressure of the vessels, the separate venous radicals may become directly diseased, particularly those surrounded by the inflamed orbital tissue, and the ethmoidal veins, covered by the diseased accessory cavity mucosa, may be subject to thrombophlebitis, and because of the vein connections mentioned may lead to embolisms, as well as to a dissemination and destruction of venous thrombus.

Kuhnt saw three cases of embolism of the *vena centralis retinae* from maxillary sinus empyema. An operation was refused, so perforce there can be no further information on the subject. The case of Fouchet showed the presence of maxillary sinus empyema, thrombophlebitis of the plexus pterygoideus, and of the plexus ophthalmicus. In cases of empyema of the frontal the following observations were made:

Zirn, thrombophlebitis of the veins of the eye and of the sinus cavernosus. Bourot-Lecard, thrombophlebitis of the veins of the eye and of the sinus cavernosus; E. Fraenkel, Macewen-Millar and Carver, thrombophlebitis of the sinus longitudinalis superior. Raymond, sphenoid empyema, thrombosis of the sinus cavernosus and of the *venae ophthalmicae*; Ortmann, thrombosis of both sinus cavernosi; Pekastovsky, thrombosis of the sinus longitudinalis; Scholz, thrombosis of the sinus caroticus and petrosus dexter; Schlagenhauer, thrombophlebitis of the sinus longitudinalis; Pauncz, throm-

bophlebitis purulenta sinus circularis Ridley et sinuum petros. inf. et sup.; Schroeder, thrombophlebitis of the sinus cavernosus and petrosus.

In cases of combined accessory cavity empyemas the following are the findings:

Duplay, empyema of the ethmoid cells and sphenoid sinus, suppurative phlebitis in most of the sinuses; Russel, empyema of the ethmoid cells and sphenoid sinus, thrombosis of the sinus cavernosus, petrosus, and the left vena ophthalmica; Vossius, empyema of the ethmoid cells and sphenoid sinus, thrombophlebitis of the vena ophthalmica superior, the vena angularis and the vena facialis anterior sinistra; Finlag, empyema of the sphenoid sinuses, and the ethmoid cells, and thrombophlebitis of the sinus cavernosus and circularis.

Concerning transmission of the inflammatory process, the congenital, physiologic and bone dehiscences caused by senile atrophy play an important part. Congenital dehiscences have been discussed in a separate chapter. Owing to the presence of congenital bone dehiscences the mucosa of the frontal and of the maxillary sinus may be in relation with the external bone periosteum and the periorbita; the mucosa of the frontal and the sphenoid sinus may touch the dura mater, the semicanalis ethmoidalis may bring the frontal and the orbital cells outwardly into relation with the periorbita, inwardly with the dura mater. The dehiscences of the canalis opticus, of the ethmoid cells and the sphenoid sinus may connect the mucosa of the cavities with the sheath of the optic nerve. The dehiscence of the lamina papyracea of the ethmoids connects the ethmoid cells with the periorbita. Concerning the dehiscences of senile atrophy, Zuckerkandl and we have observed the extreme attenuation of the walls of the sphenoid sinus, in places with holes and defects of various sizes. In a case of Zuckerkandl, the frontal communicated with the skull cavity, in the case of Merlin, the frontal did the same with the orbital cavity. In such cases periostitis, perineuritis optica and meningitis with its accompanying phenomena often occur. Dehiscences between the accessory cavities and the cranial cavity may bring about pachymeningitis, extradural abscess, rupture and cerebral abscess; dehiscences between the accessory cavities and the frontal, the cheek and fossa pterygopalatina may cause periostitis, phlegmon, abscesses, and thrombophlebitis.

Dehiscences between the accessory cavities and the orbital cavity may cause orbital emphysema, periostitis, orbital phlegmon, orbital abscess and thrombophlebitis; dehiscences of the canalis opticus may cause periostitis, perineuritis optica, neuritis and atrophy of the optic nerves. In addition to these conditions the inflammatory process may be transmitted to the periosteum, the bony walls of the accessory cavities and to the neighboring tissues, causing inflammation of the bony parts and destruction of the bones, also caries, necrosis, perforation of the individual accessory cavities, phlegmon, abscesses of the orbit and of the adjoining parts of the individual accessory cavities, accompanied by all the phenomena of disease. If the inflammation is transmitted through the bones, it may easily affect the optic nerve and its sheath in the region of the canalis opticus, invading the orbit by way of bone inflammation; periostitis appears, which transmits itself directly to the canalis opticus, causing diseased optic nerves, also orbital phlegmona, orbital abscesses, as well as pressure on the optic nerves and its vessels; furthermore, thrombophlebitis of the veins of the eyes, and its serious accompanying phenomena. Communication between the frontals and the ethmoid cells, the so-called orbital cells, may lead to a direct infection of these cavities. The thin common partition walls of the individual accessory cavities may, by means of a process of perforation, result in invasion of the neighboring accessory cavities. A rupture may therefore connect the frontals, as well as the sphenoid sinuses. In this way the diseased process may be directly transmitted from the maxillary sinus to the ethmoid cells, and to the sphenoid sinus, and continued on the same side from the ethmoid cell to the sphenoid sinus. In case of a rupture of the common wall of the ethmoid cell, the contralateral sphenoid sinus may also become diseased. These conditions may also run their course in the opposite direction. We will now discuss a few complications in connection with accessory cavities of the empyema, which not yet have been mentioned.

In case of maxillary sinus empyema, the following complications are noted: Epiphora, blepharospasmus, reflex neurosis, asthma, angina pectoris, dacryocystitis purulenta, fistulous condition of the tear-sac. In the case of Nolthenius⁸⁸ there was rupture of the maxillary sinus suppurations in the direction of

the nasolachrymal canal. In the case of Treitel⁹⁶ granulations and sequestrations of the inner wall of the maxillary sinus caused blenorrhea of the lachrymal sac and a fistulous growth. Asthenopic conditions have been observed by Gruenwald, Caldwell⁹⁶ and others. Ziem and Fromaget saw a case that was cured by operation; Kuhnt, an improvement, after treatment of iritis; Ziem and Kuhnt, improvement in sight in a case of cataract. Kuhnt saw the appearance of neuroretinitis.

Grohman⁹⁸ observed a hyperemia of the optic nerve papilla; Depaques⁹⁹ observed an obliteration of the borders of the optic nerves, with impaired sight; Courtaix and Kolarowitsch report amblyopias and amauroses of dental origin. In the cases of Koerner¹⁰⁰, LeFort¹⁰¹, Pagenstecher¹⁰², there was exophthalmus from edema of the retrobulbar cellular tissue, also impairment of sight to the extent of blindness, from pressure on the optic nerves and its vessels. There was a transient amblyopia or amaurosis in the cases of Brueck¹⁰³, Pasquier¹⁰⁴, Buzer¹⁰⁵, Galezowski¹⁰⁶ and Onodi¹⁰⁷.

The diseased condition of the bone leads to rupture, to fistulae and abscesses. Abscesses and fistulae of the hard palate were observed in the cases of Gruenwald¹⁰⁸ and Panzer¹⁰⁹, phlegmon of the fossa pterygopalatina in the cases of Tedenat¹¹⁰, also Westermayer¹¹¹. Rupture of the medial wall of the maxillary sinus into the inferior meatus was observed by Bournonville¹¹², Jansen¹¹³, Flatau¹¹⁴, and Lichtwitz¹¹⁵; into the middle meatus by Killian¹¹⁶. In the case of Dmochowski¹¹⁷ a rupture of the maxillary sinus into the sphenoid sinus took place. Rupture of the superior wall of the maxillary sinus, that is, the inferior wall of the orbital cavity, was observed in the cases of Bauby¹¹⁸, Dmochowski, Kuhnt, Salva¹¹⁹ and Fisher¹²⁰. Orbital phlegmona and orbital abscesses may appear without the presence of rupture, and as we have observed in the quoted cases, may lead to intracranial suppurations and to thrombophlebitis of the venous radicals and of the sinuses.

In cases of frontal empyema the following complications may arise: Reflex neurosis, bulbar, periorbital neuralgia and rarely lachrymal blenorrhea. According to Kuhnt and Lichtwitz, hyperemia of the papilla, filling of the veins and peripapillar retinal infiltration may exist. Kuhnt, Onodi and others noted exophthalmus and diplopia. Schmiegelow¹²⁰ and

Jacquau¹²¹ saw acute exophthalmus. Kuhnt reported opacities of the lens. According to Lapersonne¹²² transitory or permanent paralysis of the nerves of the eye muscle, edema of the retrobulbar connective tissue, orbital phlegmon, orbital abscess, periosteal inflammations and rupture of the bony walls have often been observed. Bourot, Lecard, Carver and Schroeder have observed periostitis and subperiosteal abscess of the superior orbital wall, and at the same time caries, necrosis, sequestra. Spencer-Watson¹²³ removed two sequestra of the orbital lamina of the frontal and Panas¹²⁴ one. We do not so often see changes on the anterior frontal wall; periostitis, phlegmon of the frontal derma and of the upper eyelid and abscesses have been observed by Baessler¹²⁵, Steinthal¹²⁶, Koehler¹²⁷, Roth and MacEwen-Millar, perforation by Botey¹²⁸. Perforations of the posterior frontal wall, orbital abscess, thrombophlebitis, intracranial and cerebral complications have been mentioned in the reported cases.

In cases of ethmoid empyemas, Burger¹²⁹, Caldwell¹³⁰, Hajek¹³¹, and Gruenwald observed asthenopic difficulties. Cases of retrobulbar neuritis and congestion neuritis are known; they will be discussed later. Dacryocystitis purulenta, orbital emphysema. Displacement of the eye-ball may be caused by mucocoele of the ethmoid cells, as well as by ectasia of the empyema. It is a known fact that suppurative swelling of the anterior ethmoid cells may cause displacement of the eye-ball.

Exophthalmus without impaired sight in cases of ethmoid mucocoele was observed by Hjorth¹³² and Onodi¹³³, exophthalmus with diplopia by Baurowitz¹³⁴, exophthalmus, neuritis optica by Mann¹³⁵, exophthalmus and blindness by Richet, seropurulent contents of the mucocoele with displacement of the eye-ball by Schmiegelow¹³⁶; intermittent exophthalmus, Eversbusch¹³⁷. Orbital phlegmon, orbital abscess, perforation of the ethmoid are also noted, and cases with intracranial and cerebral complications have been reported.

In cases of sphenoid sinus empyema, the following observations are noted: Cases of congestion of the papilla, retrobulbar neuritis, and atrophy of the optic nerve will be more fully discussed later. Orbital phlegmon, exophthalmus and diplopia may occur. Baumgarten¹³⁸ reports a case of oculomotor paralysis and Post¹³⁹ a cured case of necrosis of

the small wing of sphenoid with exophthalmus and permanent amaurosis.

Cases with intracranial complications have already been mentioned.

Cases of intracranial and cerebral complications with combined empyema of the accessory cavity have also been reported. Cases of neuritis and atrophy of the optic nerves will be more fully discussed later.

In the case of Sokos and Luc¹⁴⁰, diplopia occurred in empyema of the maxillary and frontal sinus. In a case of maxillary and sphenoid sinus empyema, Lapersonne observed a complete oculomotor paralysis. Ingersoll observed exophthalmus in a case of empyema of the frontal and of the ethmoid cells, and Broeckert¹⁴¹ observed dimness of the vitreous body and detachment of the retina in a case of empyema of the frontal and ethmoid cells. Hoffman¹⁴², in empyema of the frontal, the ethmoid cells and of the sphenoid sinus, found rupture into the orbit and orbital abscess, and in a case of empyema of the ethmoid cells and the sphenoid sinus exophthalmus and diplopia. F. R. Alexander¹⁴³ saw, in connection with an empyema of the sphenoid sinus and the ethmoid cells, a metastasis of the eye, and abscess of the vitreous body.

We now come to the question of atrophy of the optic nerves and canalicular neuritis brought about by diseases of the accessory cavities. As we have already mentioned, starting from the orbit the inflammation may be directly transmitted to the optic nerves, also pressure on that portion without vessels, and upon the vessels of the optic nerves may lead to impaired sight and blindness. Starting from those accessory cavities which are in close relation to the optic nerve, the inflammatory process may be directly transmitted to the walls of the canalis opticus and the sheath of the optic nerve, and that this transmission is furthered by the presence of possible bony dehiscences, is self-evident. In those cases in which the common septal wall between the canalis opticus and the accessory cavities is very thin, periostitis and disease of the bones may lead directly to perineuritis and its subsequent symptoms.

It has been possible to explain the circulatory derangements on the score of the vein connections and origin in the sinus which have been described. The postmortem findings quoted from Duplay, Horner, Panas, Rouge, Russel, Raymond,

Demarquay, Vossius, Holmes and others confirm the causal connection of sight derangement and blindness with diseases of the ethmoid cells and sphenoid sinuses. It is unfortunate that we lack the confirmation of the course of infection, the investigation of the optic nerve, its sheath, the optic vessels, the proof of the more intimate relations existing between the individual accessory cavities and the canalis opticus and between the sulcus opticus and the optic nerve and chiasma, with their possible changes. A number of observations are placed at our disposal, which lean or point towards a causal connection with positive cures of the optic nerve in connection with unfavorable course and also negative results in spite of operations.

We are acquainted with several cases in which, in spite of the presence of suppuration and destruction of the bony walls of the sphenoid sinus, the sight was not impaired. Berger and Tyrmann mention the slow erosion or wearing away of separate parts of the sphenoid body, though no derangement of sight took place, but later meningitis appeared. Baratoux¹⁴⁴ mentions a case in which a large part of the sphenoid body was thrown off through the nose, causing no disturbance whatever. Hajek saw a number of cases of syphilitic destruction of the anterior wall of the sphenoid sinus without special symptoms of any other kind. Flatau¹⁴⁵ reports twenty-six cases of empyema and caries of the sphenoid sinus, but mentions no derangement of sight. Foucher¹⁴⁶ describes the case of a girl of fifteen with necrosis of the turbinates and the sphenoid sinuses; antiluetic treatment was instituted, bone sequestra were formed and the girl died; sight disturbances were not observed. The observations of Schaffer,¹⁴⁷ Onodi,¹⁴⁸ Schmiegelow and Hoffman¹⁴⁹ also show that in many cases of diseased sphenoid sinuses no changes in the functions of the optic nerves took place. Hinkel has treated twenty cases of sphenoid sinus empyema without noticing changes in the field of vision or that they brought about diseases of the fundus of the eye. In contrast with the observations of Grünwald, Ziem,¹⁵⁰ Berger,¹⁵¹ Kuhnt and Bryan,¹⁵² Henrici and Haffner,¹⁵³ in thirty-six cases of accessory cavities diseases found normal field of vision. According to our opinion, shared by Hoffman, these facts are to be explained by reason of the observations and conditions which we have discussed.

We have spoken of those cases in which the canalis opticus and the optic nerve stand in no close relation to the ethmoid cells and the sphenoid sinuses. Furthermore, we have mentioned the observations and illustrated those preparations which show the different thicknesses of the bony substance between the sphenoid sinus and the optic nerve.

In the transmission of the inflammatory process of the sphenoid sinus, two facts play a protective and restraining part. First, we have seen how far removed the sphenoid sinus may be from the region of the optic nerve, and second, the bone substance, varying in thickness from 1 mm. to 12 mm., which separates the sphenoid sinus from the optic nerve. These facts show that empyema may cause destructive processes of the bones without sight derangements resulting.

It would lead too far afield to speak of the different causes of neuritis; we will therefore confine ourselves solely to the diseased accessory cavities in the region of the optic nerve. In this connection I must call attention to the almost mechanical theory which sought to bring about a connection between canalicular diseases of the optic nerve with diseases of the sphenoid sinus.

By reason of my investigations I have taken issue with this theory and have proven^{15*} that in many cases the sphenoid sinus has nothing at all to do with the optic nerve, and intimate relations exist only with the last posterior ethmoid cell. These facts have already been considered by Schmiegelow, Alexander and Hoffman. Pauncz,^{16*} who speaks of these facts regarding the section of the optic nerve in the canalis opticus says further that the intracranial part of the optic nerve, between the canalis opticus and chiasma, "stands almost solely in close relation with the sphenoid sinus only." This statement has been refuted by the above given findings, also his remark "that this section of the optic nerve is always separated from the sphenoid sinus by a thick bony wall." I have mentioned in my works and on this occasion must again emphasize the fact that when the ethmoid cells and the sphenoid sinus become diseased simultaneously, both cavities are to be taken into consideration; under no condition is it to be supposed that the sphenoid sinus alone is the foundation for the causal connection. Schmiegelow is correct when he brings sight derangements into causal connection with the ethmoid cells

and the sphenoid sinus, not making one sinus or the other responsible. On the contrary, Halstead¹⁵⁶ and Pollatscheck¹⁵⁷ in empyema of the ethmoid cells and of the sphenoid sinus, trace back the derangement of sight solely to a diseased sphenoid sinus, without establishing the elimination of the ethmoid cells, whereas the assumption of a causal connection with empyema of the ethmoid cells might as well have been made. According to our morphologic findings, transmission of the inflammation from the posterior ethmoid cells as well as from the sphenoid sinus, to the optic nerve, is possible, as well as circulatory disturbances.

Mendel¹⁵⁸ and Lapersonne have stated that the appearance of neuritis on one side is usually characteristic of nasal origin. Mendel finds "that almost half the cases of one-sided optic nerve inflammation are in a large measure of nasal origin." He says that unilateral choked disc is mostly an orbital affection; on both sides it is an intracranial affection.

Lapersonne emphasizes the characteristics of one-sided disease: "Optic neuritis is rarely observed in frontal sinusitis, somewhat more frequently in maxillary or ethmoid empyema, but it is especially a manifestation of sphenoid sinusitis. One main characteristic of this neuritis when purely of sinus origin is that it is unilateral. Although both nerves may be attacked in the canal by a double sphenoid sinusitis, a double edematous neuritis should make us look for an intracranial cause."

Prof. H. Sattler¹⁵⁹ in answer to my question expressed himself in the following manner: One-sided neuritis optica, or atrophy of the optic nerve, is in no way characteristic of diseases of the sphenoid and ethmoid sinuses; it may be induced by inflammation, hemorrhages, tumors, at the cerebral end of the canalis opticus and by toxic phenomena.

The report submitted by Mendel and Lapersonne cannot hold good; speaking against the assertions above given, there are, in the first place, those cases in which diminution of sight on both sides was observed in one-sided disease, furthermore the morphologic findings discovered and described by us establish the possibility of the lesion of both optic nerves and chiasm in diseases of one posterior ethmoid cell or a sphenoid sinus on one side. One fact presented by Mendel and Lapersonne we are able to confirm, namely, that in most cases heretofore under observation, a unilateral disease of the accessory

cavities brought about the sight derangements and blindness. The number of observed cases of suppuration of the accessory cavities on both sides is considerably smaller, as well as the number of cases of contralateral and double-sided sight derangements in one-sided diseases of the accessory cavities.

Before considering these cases collectively, we wish to call attention to an etiologic point and this is the artificial impairment of sight caused by an operation. This occurred in a case observed and published by Hirschberg¹⁰⁰ in which, after operation for empyema of the right maxillary, ethmoid and sphenoid sinuses, a superior, partial immobility of the pupil ensued. This traumatic lesion of the optic nerve was brought about by the clearing out of the posterior ethmoid cells and the sphenoid sinuses through the maxillary. The topographic relations discussed explain these artificial injuries, particularly the varying course of the optic nerve along the posterior ethmoid cell and the sphenoid sinus. A glance at figures 8, 9 and 27 will clearly explain the origin of a traumatic lesion of the optic nerve in cases of cleansing out of the cavities in question.

Concerning disease of the accessory cavities on one side with unilateral sight derangements and blindness of the same side, we will mention the cases already noted with post-mortem findings of Duplay, Horner, Panas, Rouge, Russell, Raymond, Demarquay, Leber and Holmes. Cases are mentioned in which accurate data concerning nose-findings, diagnosis and operative measures are lacking. Berger reports two cases of neuritis ending with amaurosis. Grossman¹⁰¹ thought that in four cases there was an etiologic connection between sight impairment and the diseased sphenoid sinus. Snellen¹⁰² traced two cases of optic nerve atrophy back to sphenoid sinus empyemas. Knapp¹⁰³ assumes that in one case a sphenoid sinus empyema led to changes of the papilla.

Hirschman writes me of a case in which neither a necropsy nor an operation was permitted and as the cause of a temporal hemianopsia, the atrophic discoloration of the temporal half of the left papilla and the paleness of the same half on the right side, assumes a diseased sphenoid sinus or a tumor in the region of the sella turcica.

In the cases of Lapersonne and Gronbäk¹⁰⁴ the operation was not successful. In the case of Gronbäk the amaurosis

with empyema of the ethmoid cells remained unchanged. Operations in Lapersonne's three cases did not influence the unfavorable progress of the neuritis optica. In the case of Post, necrosis of the smaller wing of the sphenoid caused exophthalmus and amaurosis. After the extraction of the necrotic bone the eye resumed its normal position, the amaurosis, however, remained. In the case of Risley¹⁶⁵ empyema of the ethmoid cell and the sphenoid sinus caused blindness of the eye.

We will now discuss those cases in which sight derangements on one side with diseased accessory cavities were observed and cured. But we must touch first on the case of Coppez and Lor,¹⁶⁶ who observed in a girl 20 years old, a neuritis optica of one side which was cured in spite of the fact that the girl would allow no surgical operation of her chronic sphenoid sinus empyema. Mendel also mentions cases of so-called retrobulbar neuritis which healed spontaneously or were cured by simple treatments. In the case of Coppez,¹⁶⁷ the bulb was turned outward with slight exophthalmus, derangement of vision, left opticus indistinct, suppuration of sphenoid sinus and sequestrum 1 cm. long. Mendel describes a case of unilateral retrobulbar neuritis which, after the suppurating ethmoid cells were opened, showed distinct improvement. In the case of Bergmeister-Hajek,¹⁶⁸ one-sided hyperemic neuritis, empyema of the ethmoid labyrinth resulted in cure. In the case of Fliess¹⁶⁹ retrobulbar neuritis, sphenoid sinus empyema of the right side, treatment was crowned by success. In the case of Lor¹⁷⁰ sphenoid sinus empyema and neuritis, with sudden blindness, operation, cure. Nuel¹⁷¹ observed three similar cases of neuritis or neuroretinitis. In the case of Hoffman¹⁷² treatment of empyema of the sphenoid sinus and the ethmoid cells was successful. In the case of Holmes¹⁷³ loss of the eyesight of the left eye, empyema of the left sphenoid sinus, opening, cure effected. In the case of Pauncz¹⁷⁴ the papillitis nervi optici showed decided improvement after the ethmoid cells were opened and washed out. In the case of Schmiegelow¹⁷⁵ the eyesight showed decided improvement after an operation for empyema of the sphenoid sinus and the last ethmoid cells. In the case of Fuchs-Hajek¹⁷⁶ cure of a retrobulbar neuritis with central scotoma was effected after the removal of the polypoid de-

generated mucosa of the left posterior ethmoid cells and of the sphenoid sinus.

Concerning sight derangements of both sides and the diseased accessory cavities of both sides, we have but few cases at our command. Mendel mentions a case of double-sided optic nerve disease, caused by empyema of the ethmoid cells, giving no further detail than his source (English literature) on the subject. We saw a case of multiple accessory cavity suppuration and double atrophy of the optic nerves. In the case of Pollatschek¹⁷⁷ blindness on both sides from empyema of the sphenoid sinuses, was assumed. In the discussion we doubted the causal connection.¹⁷⁸ In the case of F. R. Alexander¹⁷⁹ suppuration of the left sphenoid sinus and the right ethmoid cells, papillitis on both sides; after opening the sinuses, a cure was effected. In the case of Schmiegelow¹⁸⁰ double empyema of the sphenoid sinus and the ethmoid cells, double neuritis, an operation, and marked improvement of sight. In the case of Delneuveville¹⁸¹ sight derangements on both sides, pus in the middle meatus. There was probably diseased sphenoid sinuses with or without conjunction of the ethmoid cells. After a month of purely medicinal treatment a cure was effected. The described intimate relation of the canalis opticus, sulcus opticus, the optic nerve and the chiasm to the posterior ethmoid cell, to the sphenoid sinus on one side, and also on both sides, the varying course of the canalis opticus with the optic nerve in the sinuses, the ethmoidal veins often lying free in the semicanalis ethmoidalis, the walls, thin as tissue paper, of the bone partitions, all these give an anatomic foundation for the explanation of the given observations.

For the origin and explanation of contralateral and double-sided sight derangements and blindness, with one-sided diseases of the accessory cavities, the etiologic causes already mentioned and the anatomic foundation mentioned by us may be useful. Our observations bear on the following cases:

1. The left posterior ethmoid cell constitutes the medial wall of the right canalis opticus.
2. The right posterior ethmoid cell constitutes the inferior and medial wall of the canalis opticus on both sides, and the wall of the entire sulcus opticus.

3. The right posterior ethmoid cell constitutes the wall of the entire sulcus opticus.

4. The right posterior ethmoid cell constitutes the wall of the right third and the middle third of the sulcus opticus.

5. The left sphenoid sinus constitutes the inferior wall of the right canalis opticus.

6. The right sphenoid sinus borders medially only the left optic nerve.

7. The left sphenoid sinus constitutes the inferior wall of the right canalis opticus, and the wall of the right third and the middle third of the sulcus opticus.

8. The left sphenoid sinus constitutes the inferior and medial wall of the canalis opticus on both sides, and the wall of the entire sulcus opticus.

9. The same morphologic relation with this difference, that only a small part of the right inferior and medial wall of the canalis opticus is constituted by the left sphenoid sinus; the larger part is constituted by the right posterior ethmoid cell.

10. The left sphenoid sinus constitutes the inferior and medial wall of the left canalis opticus, and the inferior wall of the right canalis opticus, also the wall of the entire sulcus opticus.

11. The right sphenoid sinus constitutes the wall of the middle third of the sulcus opticus.

The clinical material known to us has not been abundant. In the case of Wohlmuth¹⁸² the right frontal and the orbital edge show signs of injury, bruise of the back of the head blindness of left eye confirmed. Berlin¹⁸³ assumed that the fracture of the right orbital roof extended along the left side, in this way striking the left canalis opticus or the indirect fracture was due to the injury of the occiput.

In the case of Freudenthal¹⁸⁸ after a Killian operation on the right side of the frontal, the left eye became blind and six weeks afterwards atrophy of the optic nerves was evident. The ophthalmologists, Oppenheimer and May, considered that in consequence of the Killian operation, there was an indirect fracture in the region of the left canalis opticus. Freudenthal, having no ground for other causes, also assumed this to be the case. In the case of Laas and Lewy, Lewy removed a piece of nasal septum on the left side 4 cm. long, 2½ cm. wide, which lay obliquely from below backwards. A quarter

of an hour after the operation there were scintillations before the right eye and lessened sight. Upon examination Laas found almost total defect of the superior field of vision, normal fundus, the right papilla beginning to become pale; in two weeks the right opticus was clear white. Laas and Lewy were of the opinion that there was a fracture of the right canalis opticus. By correspondence I explained this case on the assumption that there was a fracture of the right canalis opticus. Pointing to this is the sudden appearance of sight impairment; my anatomic findings, which show the free course of the canalis opticus in the sphenoid sinus and the rear ethmoid cell, also those preparations which in the Atlas figures 14, 15, 67, 72, 73 and 91 illustrate, and which show the spreading out of the sphenoid sinuses between the lamellae of the nasal septum; furthermore, the spreading out of the sphenoid sinus, and of the posterior ethmoid cell from one side over the partition wall to the other side. These findings may explain an indirect fracture of the canalis opticus in the given case.¹⁸⁵ As regards the cases of Wohlmuth, Freudenthal, and Laas and Lewy, it is impossible to deny the probable existence of an indirect contralateral fracture, in the region of the canalis opticus, the foramen opticum, and the sulcus opticus, considering the fact that indirect fractures of the canalis opticus on the same side as the injuries have often been observed, and cases under our notice have shown that the posterior ethmoid cell as well as the sphenoid sinus may be separated from the opposite canalis opticus by a bony wall of paper-like thinness. Hölder has observed fifty-three cases of fracture of the walls of the canalis opticus. Should an indirect fracture arise, in view of the existing relations observed by us, its position must be contralateral. But if we give our closer attention to the Freudenthal case, two facts must give food for reflection: first, in spite of numerous operations on the skull, done with hammer and chisel, the history of not a single case is known; second, the patient's frontal was first opened according to Kuhnt's method, and the assumed indirect contralateral fracture must have, in consequence, appeared after the subsequent demolition of the inferior frontal wall. In all operations performed according to the Killian method, in which the use of hammer and chisel is more general, than in this case, traumatic lesion of the optic

nerve on the same side or on the contralateral has not been observed. We could not forego these few remarks, although in consequence of our described investigations, we declared an indirect contralateral fracture of the canalis opticus possible. In the case of Halstead¹⁸⁶ there were empyema of the maxillary sinus, the ethmoid cells and sphenoid sinus of the right side with blindness of the left eye. Halstead thought that there was a rupture of the empyema of the right sphenoid sinus into the left sphenoid sinus, in consequence of which an old left-sided neuritis responded. Brown thought that there was exudation in the left optic sheath; half a year after the operation the left eye was normal. Rhinoscopically the rupture of the empyema of the right sphenoid sinus into the left one was not proven, and the ophthalmoscopic findings could not establish the presence of an old neuritis. We must mention the existence of an empyema of the ethmoid bone, and in consequence it may be assumed that there is a causal connection as well with the empyema of the ethmoid cells. According to our morphologic findings, transmission of the inflammation from the posterior ethmoid cell, as well as the sphenoid sinus to the contralateral optic nerve is possible, as well as a circulatory disturbance; without hypotheses this is given as the reason for contralateral sight impairments. In the case of Gregg and Hay¹⁸⁷ with empyema of the right ethmoid cells there occurred hemianopsia bitemporalis, and paralysis of the associated movements. Cure was due to operative measures. They took the empyema to be the direct cause of the lesion of the chiasm and the crossing of the fibres of the Gudden commissure caused the associated derangements. Our morphologic findings make lesion of the chiasm possible from diseases of the posterior ethmoid cells as well as the sphenoid sinuses. The part which the Gudden commissure plays in the associated movements comes within the province of the neurologist and ophthalmologist. In the case of Pollatscheck,¹⁸⁸ with empyema of the left posterior ethmoid cells and sphenoid sinus, papillitis on both sides, a cure was effected by operation. He thought that there was bony dehiscence of the sphenoid sinus on only one or both sides, and that the resultant periostitis extended per continuitatem to the optic nerve on the other side. Concerning this case, in his description he says that upon opening

the posterior ethmoid cells a great deal of fetid pus was evacuated, but on opening the sphenoid little pus came out; the explanation rests on the hypothesis of a sphenoid sinus having bony dehiscences on one or both sides, and of a periostitis, with transmission to the optic nerve. Data are completely lacking concerning the extent of the opened posterior ethmoid cells, and the sphenoid sinus, and the reasons for exenterating the posterior ethmoid cells. Our morphologic observations admit the possibility of a causal connection with the diseased posterior ethmoid cells as well as diseases of the sphenoid sinus; still the question arises, as to the accidental spontaneous appearance of acute one-sided empyema and papillitis of both sides. In the case of Polyak¹⁸⁹ a bone cyst on the left side and latent multiple accessory cavity empyemas were accompanied by exophthalmus and atrophia optica on both sides; a year earlier there was disease of the right eye. He considered that the pus from the bone cyst forced its way successively into the left accessory cavities, then into those of the right side, and finding no way of flowing off, caused the dilation of the accessory cavities and pressure. In this way he explains the exophthalmus and atrophy of the optic nerves on both sides. We believe that Polyak's explanation does not hold good. The observations made by Goldzieher and myself, which had for their object a more plausible interpretation, we shall leave unmentioned, as we have recently learned that a necroscopic investigation is under way, the result of which is finally to settle this point. In the case of Gutmann,¹⁹⁰ empyema of the left sphenoid sinus was associated with blindness on the right side. He believes that a transmission to the ethmoid cells and the sphenoid sinus caused an amaurosis.

In our case, besides left-sided empyema of the posterior ethmoid cells, and the sphenoid sinus, earlier atrophia optica of the right side, neuritis optica with hemianopsia temporalis of the left side, also existed. After an operation, the eye showed a slight improvement, vision 6/15 to 6/10. Six years before a large number of polypi had been taken out of the left nasal cavity, then there was a suppurative fetid discharge on the left side, with constant headaches; in the course of two months the right eye became blind; three months later the sight of the left eye was impaired, and investigation showed a temporal

hemianopsia. Slight improvement only was reported after operation. Lues was not indicated, as antiluetic treatment proved negative. We illustrate in figure 33 the temporal hemianopsia of the left eye. The patient did not remain longer under observation, so I cannot report further as to the fate of the left eye. Prof. Szily left the case to me, and we decided that the causal connection was entirely possible; there seemed no reason for any other construction, besides, it corresponds to our morphologic findings. I possess a preparation, in which the left sphenoid sinus only constitutes the wall of the right canalis opticus, and the wall of the middle third of the sulcus opticus. A right-sided atrophy, and left-sided temporal hemianopsia would be explained anatomically by a lesion of the right optic nerve and of the chiasm by a diseased left sphenoid sinus. The accidental appearance of both diseases spontaneously can only be explained by necroscopic investigation.

Concerning contralateral sight impairment, we have at our command no postmortem findings. The morphologic relations we have observed constitute the anatomic foundation explaining the lesion of the contralateral optic nerve and the chiasm, furthermore, that of the chiasm or both optic nerves in a disease of one side of a posterior ethmoid cell, or of a sphenoid sinus.

In regard to the therapy of diseases of the individual cavities, I refer to our work,¹⁹¹ in which I have described in detail on pages 236 to 401 the treatment of individual cavities, and the different courses of procedure. In addition, on pages 382 to 401, I have devoted special chapters to the indications for interference in the treatment of the complications of the individual accessory cavities.

I wish to make a supplementary statement. I have, in general, called attention to the latent foci, and their explorations, but must emphasize that fact that in those cases of sight derangement, in which the oculist cannot confirm the cause, and the suspicion of nasal connection exists, even though the findings be negative, it seems well to take explorative measures, and to seek the cause, whether or not the presence of latent foci is suspected. The good success of Schmiegelow and others strengthen us in our belief. I have never failed to use this test in cases given over to our care, and if I was not always

successful in locating latent foci, at least I felt nothing had been neglected. I herewith close my clinical observations.

The causal connection of a one-sided, double-sided or contralateral sight impairment and blindness in diseases of the accessory cavities, particularly those of posterior ethmoid cells and the sphenoid sinus, is conclusively proven; the anatomic foundation of this theory is sufficiently established by reason of my morphologic findings; they allow of every natural conclusion, without making use of any forced or improbable hypotheses. An exact pathologico-anatomical foundation is still to be created.

I am convinced that a complete elaboration of this theory would be made possible by exact clinical observations, and through necroscopic investigations, that mutual rhinologic and ophthalmologic researches will close this important and interesting chapter of medicine.

REFERENCES.

1. Berger and Tyrmann. Die Krankheiten der Keilbeinhöhle und des Siebbeinlabyrinthes, 1886
2. Zuckerkandl. Anatomie der Nasenhöhle, 1882, 1893.
3. Douglas. Monatsschrift für Ohren- und Kehlkopfkrankheiten, 1897.
4. Onodi. Die Nebenhöhlen der Nase, 1905.
5. Onodi. Die Dehiscenzen der Nebenhöhlen der Nase. Archiv. für Laryngologie, Bd. xv.
6. Zuckerkandl. Anatomie der Nasenhöhle, 1893.
7. Merlin. Berichte des Naturwissenschaftlich-medizinischen Vereines in Innsbruck, 1884-1885, 1885-1886.
8. Zuckerkandl and Helly. Deutsche Zeitschrift für Chirurgie, Bd. III.
9. Denouvilliers and Gosselin. Compend. de chir. prat., 1852.
10. Winkler. Archiv. für Laryngologie, 1894.
11. Killian. Münchener medizinische Wochenschrift, 1897.
12. Hyrtl. Vergangenheit und Gegenwart des Museums für menschliche Anatomie, 1869.
13. Spee. Bardelebens Handbuch der Anatomie, 1896.
14. Gallmaerts. Annal. d. Ocul., 1900.
15. Holmes. Arch. of Ophthalm., 1896.
16. Onodi. Archiv. für Laryngologie, Bd. xiv und xv.
17. Onodi. Archiv. für Laryngologie, Bd. xl.
18. Onodi. Archiv. für Laryngologie, Bd. xv.
19. Bering and Wicherikewicz. Berliner klinische Wochenschrift, 1882.
20. Schmidt-Rimpler. Archiv. für Laryngologie, Bd. xvii.
21. Onodi. Archiv. für Laryngologie, Bd. xvii.
22. Virchow. Krankhafte Geschwülste.
23. Manz. Heidelberger Berichte, 1887.

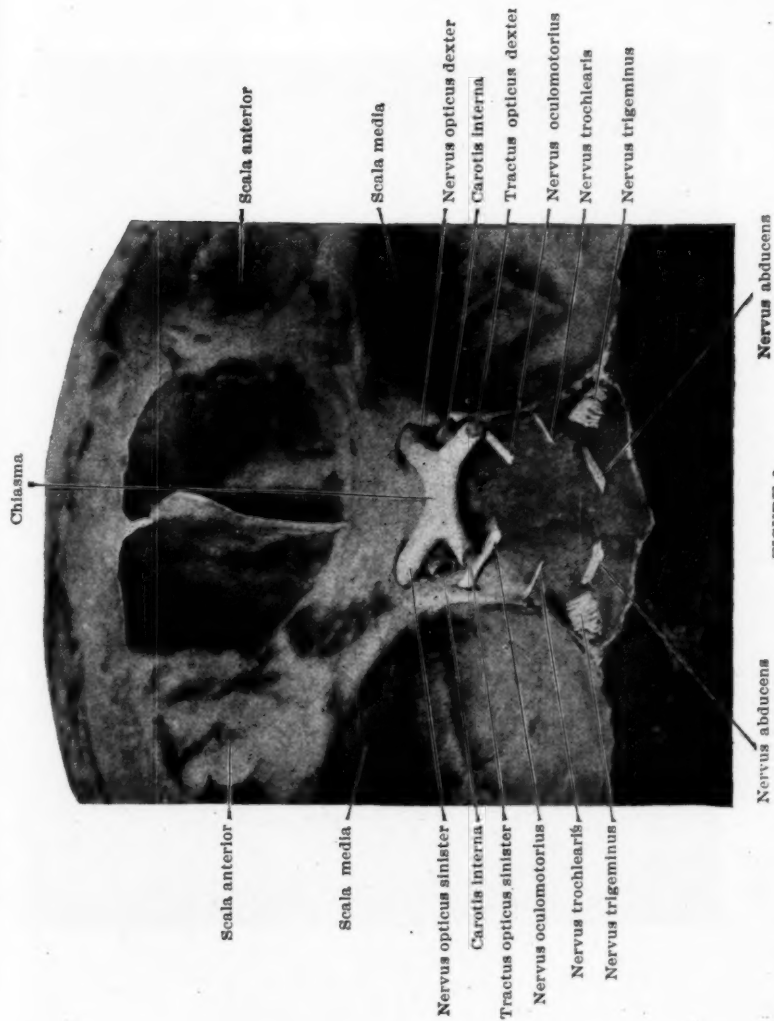
24. Enslin. Graefes Archiv für Ophthalmologie, Bd. lviii.
25. Goldzieher. Budapesti orvosi ujság, Szemeszet, 1904.
26. Hölder. Berlin, Graefe-Sämisch Handbuch.
27. Ortmann. Virchow's Archiv, Bd. cxx.
28. Hajek. Archiv. für Laryngologie, Bd. xviii.
29. Westenhöfer. Berliner klinische Wochenschrift, 1905.
30. Westenhöfer. Deutsche medizinische Wochenschrift, 1906.
31. Foucher. Courtaix, Recherches cliniques sur les relations patholog. entre l'oeil et les dents, Paris, 1895.
32. Mair. Edinburgh Med. Journ., 1866.
33. Panas. Archiv. d' Ophthalmol., 1885.
34. Dmochowsky. Archiv. für Laryngologie, 1895, Bd. iii.
35. Westermayer. Münchener medizinische Wochenschrift, 1895.
36. Bousquet. Progrés Medical, 1877.
37. Koehler. Charite-Annalen, 1892.
38. Schindler. Archives de méd. et de pharm.-militaires, 1892.
39. Zirm. Wiener medizinische Wochenschrift, 1892.
40. Huguénin. Korrespondenzblatt der Schweizer Aerzte, 1882.
41. Müller. Wiener klinische Wochenschrift, 1895.
42. Paulsen. Hospitals Tidende, 1861.
43. Knapp. Archiv. für Augenhellkunde, 1880.
44. Bourat-Lécard. Bordeaux méd., 1875.
45. Redtenbacher. Wiener medizinische Blätter, 1892.
46. Lennox-Browne. Journal of Laryngol., Vol. vii.
47. Hoppe. Klinische Monatsblätter für Augenhellkunde, 1893.
48. Krecke. Münchener medizinische Wochenschrift, 1894.
49. Wallenberg. Neurologisches Zentralblatt, 1895.
50. Silex. Berliner klinische Wochenschrift, 1896.
51. Milligan. The Medical Chronicle, 1899.
52. Fränkel. Virchow's Archiv, Bd. cxliii.
53. Macewen-Millar-Killian. Verein süddeutscher Laryngologen, 1900.
54. Roth. Wiener klinische Wochenschrift, 1899.
55. Carver. British Med. Journ., 1883.
56. Cholle. Archives internat. de larvng., 1906.
57. Begbie. Med. Times and Gaz., 1852.
58. Schäffer. Prager medizinisches Wochenschrift, 1883.
59. Pauncz. Archiv. für Laryngologie, Bd. xlii.
60. Jacobasch. Berliner klinische Wochenschrift, 1875.
61. Ogston. British Med. Journ., 1885.
62. Warner. British Med. Journ., 1885.
63. Ewald. Deutsche medizinische Wochenschrift, 1890.
64. Trousseau. Clinique médicale, Deutsche, 2. Aufl., 1866.
65. Hajek. Archiv. für Laryngologie, Bd. xviii.
66. Horner. Klinische Monatsblätter für Augenhellkunde, 1863.
67. Panas. Soc. de Chir. de Paris, 1873.
68. Rouge. L' Union médic., 1872.
69. Raymond. Bull. Soc. Anatom., Paris, 1895.
70. Grünwald. Die Lehre von den Naseneiterungen, 1893.
71. Vince. Tod, La Clinique, 1900.
72. Thiroloix. Bull. Soc. Anat., 1892.
73. Flatau. Nasenkrankheiten, etc., 1895.
74. Zöckendorfer. Prager medizinische Wochenschrift, 1893.
75. Pekastovsky. Internationales Zentralblatt. für Laryngologie. Bd. x.
76. Scholz. Berliner klinische Wochenschrift, 1892.

77. Schlagenhauser. Wiener klinische Wochenschrift, 1899.
78. Pauncz. Zeitschrift für Augenheilkunde, 1906.
79. Halász. Orvosi Hetilap, 1904.
80. Schroeder. Zeitschrift für Ohrenheilkunde, 1906.
81. Weichselbaum. Wiener medizinische Wochenschrift, 1890.
82. Pauncz. Orvosi Hetilap.
83. Hansberg. Zeitschrift für Ohrenheilkunde, Bd. xlv.
84. Duplay. Arch. gén. de méd., 1874.
85. Russel. Med. Times and Gaz., 1878.
86. Vossius. Zeitschrift für Augenheilkunde, 1900.
87. Holmes. Archives of Ophthalmology, 1886.
88. Demarquay. M. Mackenzie, Die Krankheiten der Nase, etc., 1884.
89. Panse. Archiv. für Laryngologie, I. c.
90. Leber. Archiv. für Laryngologie, Bd. xvii.
91. Finlag. Monatsschrift für Ohrenheilkunde, 1905.
92. Kuhnt. Entzündliche Erkrankungen der Stirnhöhle, 1895.
93. Kuhnt und Gurwitsch. Graefes Archiv, Bd. xxix.
94. Krauss. Münchener medizinische Wochenschrift, 1907.
95. Nolthenius. Monatsschrift für Ohrenheilkunde, 1895.
96. Treitel. Verhandlungen der Laryngologischen Gesellschaft, Berlin, 1900.
97. Caldwell. Med. Journ. of New York, 1898.
98. Grossmann. Weinlechner, Bericht. der K. K. Krankenanstalt Rudolfstiftung, 1875.
99. Depaques. Bull. de la soc. d'ophth. de Paris, 1895.
100. Körner. Verhandlungen der Deutschen odontologischen Gesellschaft, Bd. vii.
101. Le Fort. France méd., 1876.
102. Pagenstecher. Archiv. für Augenheilkunde, 1884.
103. Brück. Wochenschrift für die gesamte Heilkunde, Berlin, 1851.
104. Pasquier. Lanc. franc., 1839.
105. Buzer. Berliner klinische Wochenschrift, 1868.
106. Galezowski. Archives inter. de Laryng., 1905.
107. Onodi. Revue hebdomadaire de laryngol., etc., 1895.
108. Grünwald. Lehre von der Naseneiterung, 2. Aufl.
109. Panzer. Wiener klinische Wochenschrift, 1896.
110. Tedenat. Soleville, Thèse de Montpellier, 1890.
111. Westermayer. Münchener medizinische Wochenschrift, 1895.
112. Bournonville. Zentralblatt für Chirurgie, 1885.
113. Jansen. Archiv. für Laryngologie, Bd. I.
114. Flatau. Rev. int. de rhin., 1893.
115. Lichtwitz. Annal. de malad. d'or., 1896.
116. Killian. Heymann's Handbuch, Bd. III.
117. Dmochowski. Archiv. für Laryngologie, Bd. III.
118. Bauby. Arch. d. ophthalm., 1897.
119. Salva. Thèse de Paris, 1895.
120. Schmiegelow. Archiv. für Laryngologie, Bd. xvi.
121. Jacqueau. Internationales Zentralblatt für Laryngologie, Bd. xvi.
122. Lapersonne. Bull. de la société franc. d'ophth., 1902.
123. Spencer-Watson. Diseases of the Nose and Its Accessory Cavities.
124. Panas. Progrès méd., 1887.
125. Bäumlér. Kongress für innere medicin, 1890.

126. Steintal. Medizinisches Korrespondenzblatt der Württembergischen ärztlichen Landesvereine, 1891.
127. Köhler. Charité Annal., 1891.
128. Botey. Revue de laryng., 1897.
129. Burger. Zentralblatt für Laryngologie, Bd. xi.
130. Caldwell. Zentralblatt für Laryngologie, Bd. x.
131. Hajek. Erkrankungen der Nasenhöhlen der Nase, 1903.
132. Hjorth. Schmiegelow, Archiv. für Laryngologie, Bd. xv.
133. Onodi. Archiv. für Laryngologie, Bd. xvii.
134. Baurowitz. Archiv. für Laryngologie, Bd. xii.
135. Mann. Verhandlungen der Deutschen otologischen Gesellschaft, 1901.
136. Schmiegelow. Archiv. für Laryngologie, Bd. xv.
137. Eversbusch. Graete-Sämisch Handbuch, 2 Aufl.
138. Baumgarten. Orvosi Hetilap, 1903.
139. Post. Lancet, 1882.
140. Sakos und Luc. Revue de laryngol., 1899.
141. Broeckart. Revue de laryngol., 1901.
142. Hoffmann. Zeitschrift für Augenheilkunde, 1906.
143. Alexander. Verhandlungen der Deutschen otologischen Gesellschaft, 1905.
144. Baratoux. Arch. ital. di laryng., 1883.
145. Flatau. Zentralblatt für Laryngologie, 1894-1895.
146. Foucher. Zentralblatt für Laryngologie, 1894-1895.
147. Schäffer. Deutsche medizinische Wochenschrift, 1892.
148. Onodi. Archiv. für Laryngologie, l. c.
149. Hinkel. American Laryng. Assoc., 1902.
150. Ziem. Berliner klinische Wochenschrift, 1888.
151. Berger. Rapports entre les malad. des yeux, etc., 1892.
152. Bryan. American Laryng. Assoc., 1895.
153. Häffner. Münchener medizinische Wochenschrift, 1904.
154. Onodi. Zeitschrift für Augenheilkunde l. c.; Archiv für Laryngologie, l. c.
155. Pauncz. Archiv. für Augenheilkunde, Bd. lli.
156. Halstead. American Laryng., Rhin. and Otol. Soc., 1901.
157. Pollatschek. Zentralblatt für Laryngologie, Bd. xxii.
158. Mendel. Zentralblatt für Augenheilkunde.
159. Sattler. Onodi, Archiv. für Laryngologie, Bd. xvi.
160. Hirschberg. Zentralblatt für Augenheilkunde.
161. Grossmann. Allgemeine Wiener medizinische Zeitung, 1893.
162. Snellen. Med. Tidschr. vor Geneesk., 1894.
163. Knapp. Zeitschrift für Ohrenheilkunde, 1894.
164. Gronbäk. Verhandlungen des Dänischen oto.-laryngologischen Vereines, 1904.
165. Risley. Internationales Zentralblatt für Laryngologie, 1905.
166. Coppez und Lor. La Presse méd. Belge., 1900.
167. Coppez. Ophthalmologische klinik, 1902.
168. Hajek. Die Erkrankungen der Nebenhöhlen der Nase, 1903.
169. Fliess. Wiener klinische Rundschau, 1895.
170. Lor. Schmiegelow, Archiv. für Laryngologie, Bd. xv.
171. Nuel. Schmiegelow, Archiv. für Laryngologie, Bd. xv.
172. Hoffmann. Verhandlungen der Deutschen otologischen Gesellschaft, 1897.
173. Holmes. Archives of Ophthalmol., 1896.
174. Pauncz. Zentralblatt für Laryngologie, Bd. xxii.
175. Schmiegelow. Archiv. für Laryngologie, Bd. xviii.

176. Fuchs-Hajek. Fuchs, Lehrbuch der Augenheilkunde, 1905.
177. Pollatschek. Ovorsí Hetilap.
178. Pottalschek. Ovorsí Hetilap.
179. Alexander. Verhandlungen der Deutschen otologischen Gesellschaft, 1905.
180. Schmiegelow. Archiv. für Laryngologie, Bd. xviii.
181. Delneuvillle. La Presse oto-laryngologique Belge, 1906.
182. Wohlmuth. Berlin, Graefe-Sämisch, 1. Aufl.
183. Berlin. Idem.
184. Freudenthal. Archives internat. Chaveau et St. Hilaire, Paris, 1905.
185. Laas reported this case in the February meeting of the Berliner ophthalmologischen gesellschaft.
186. Halstead. Americ. Laryng., Rhin. and Otol Sol., 1901.
187. Glegg und Hay. Archiv. für Laryngologie, Bd. xvi.
188. Pollatschek. Zentralblatt für Laryngologie, Bd. xxii.
189. Polyák. Archiv. für Laryngologie, Bd. xv.
190. Gutmann. Zeitschrift für Augenheilkunde, 1906.
- 191 Onodi und Rosenberg. Die Behandlung der Krankheiten der Nase und des Nasen-Rachenraumes, 1906.

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Dura mater

Lobus frontalis

Nervus opticus

Carotis interna

Sinus sphenoidalis

Recessus orbitalis

Sinus frontalis

Cavum narium

Sinus maxillaris

X X X X X X X X X X Cellulae ethmoidales anteriores et posteriores.
FIGURES 3 and 19.

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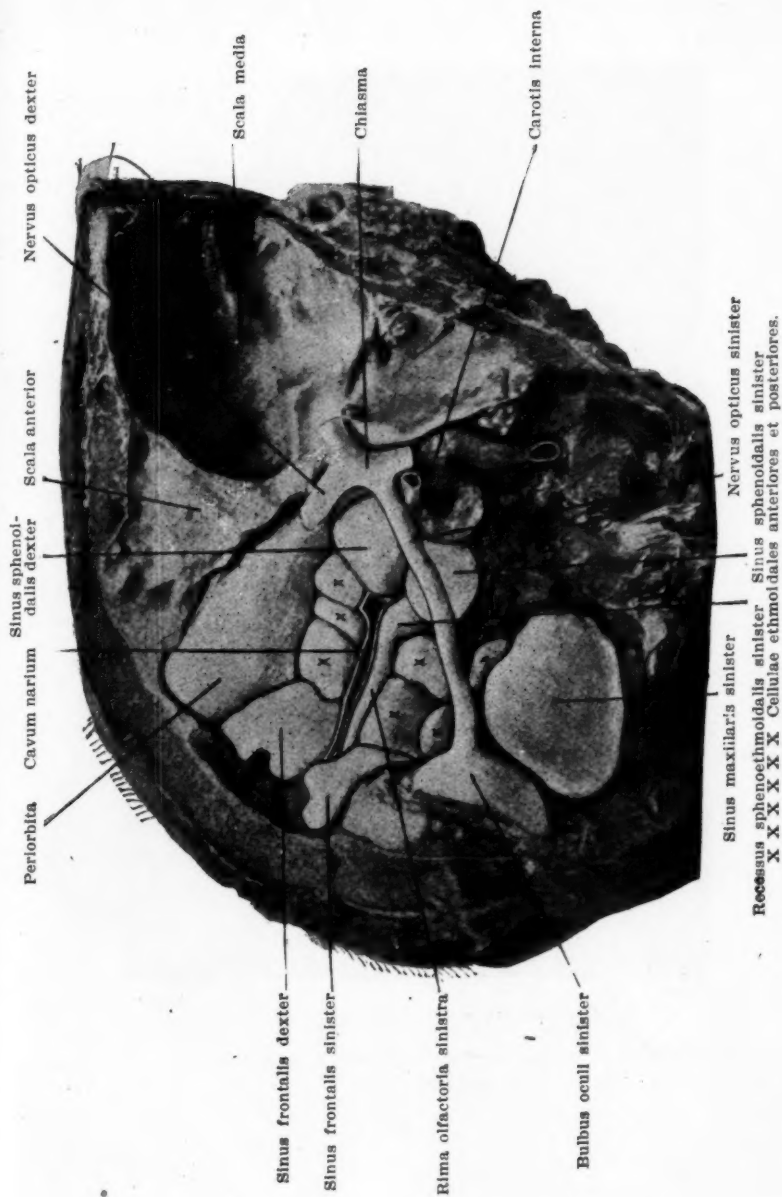
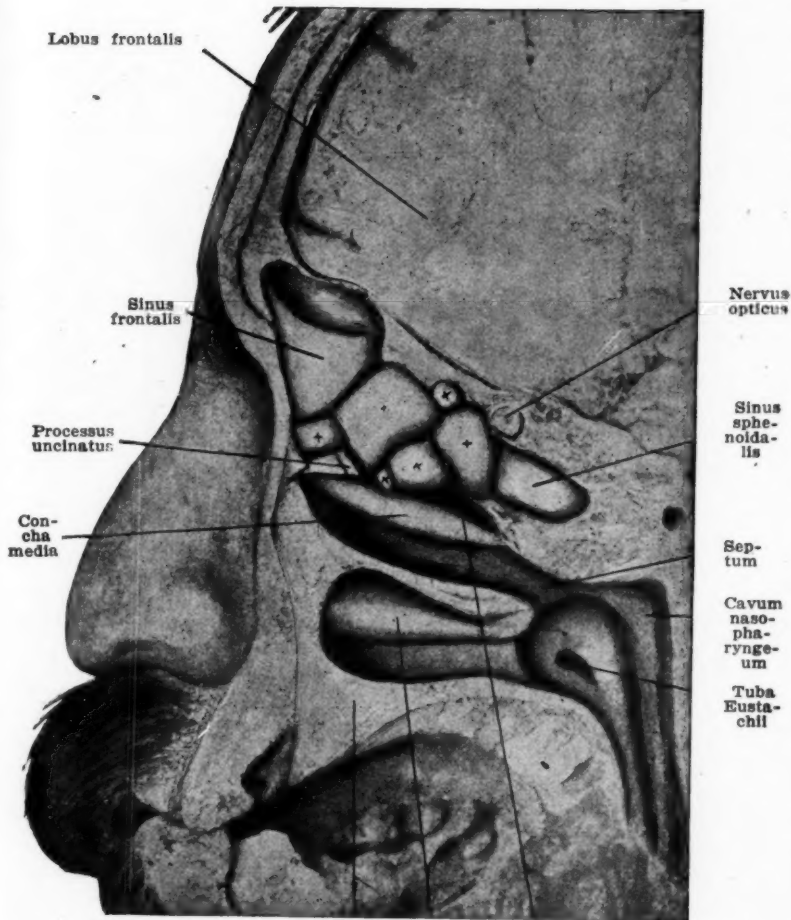


FIGURE 4.

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Palatum Concha Meatus narium
 inferior superior

X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 5.

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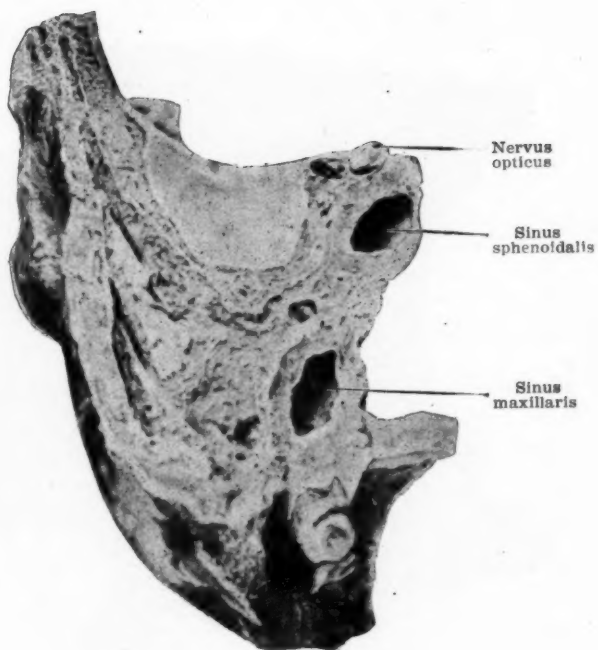


FIGURE 6.

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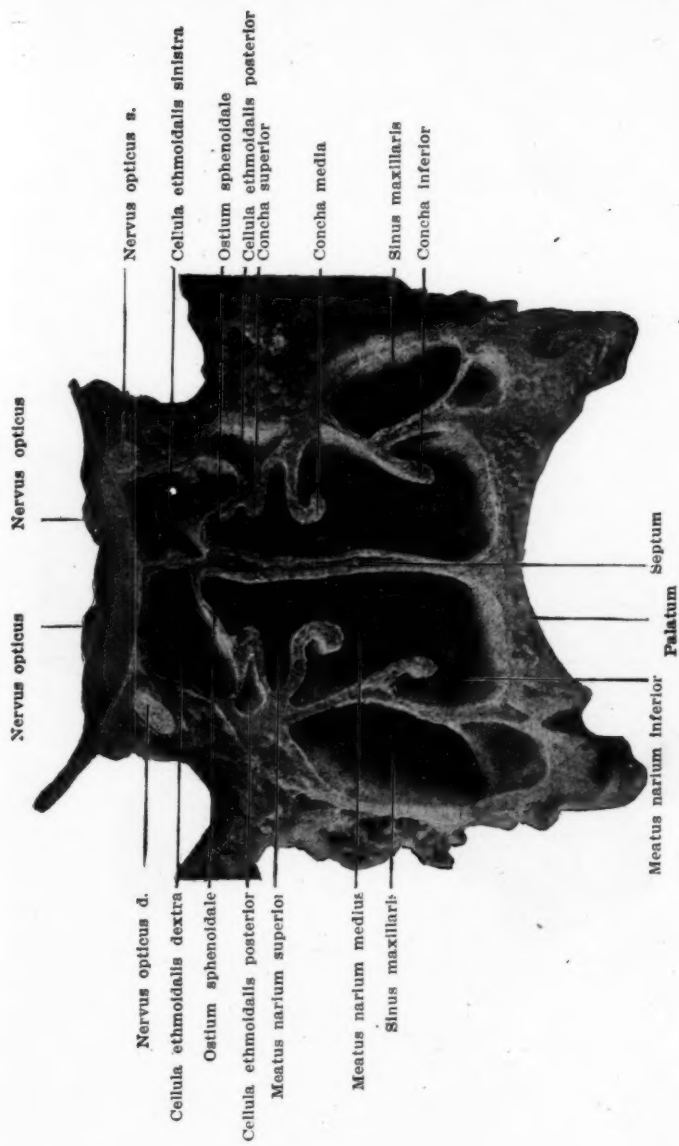


FIGURE 7.

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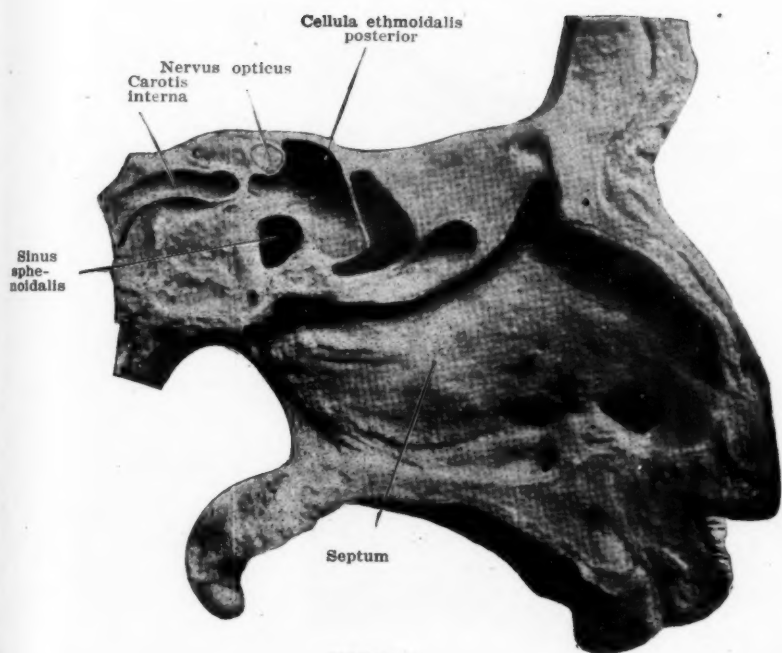


FIGURE 8.

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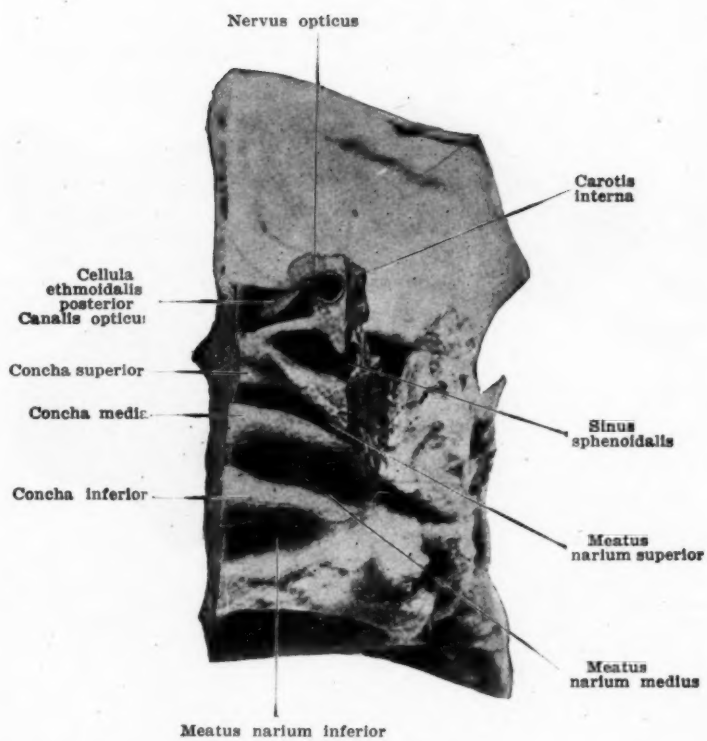


FIGURE 9.

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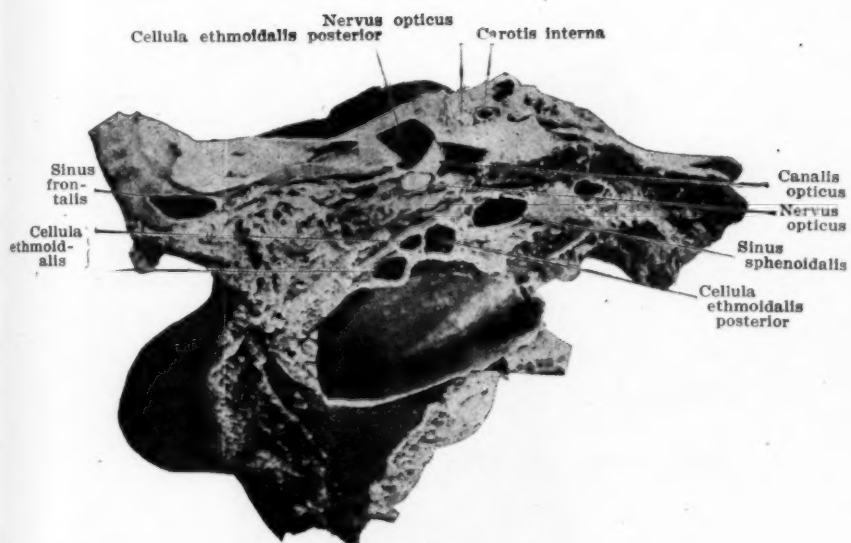


FIGURE 10.

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FIGURE 11.

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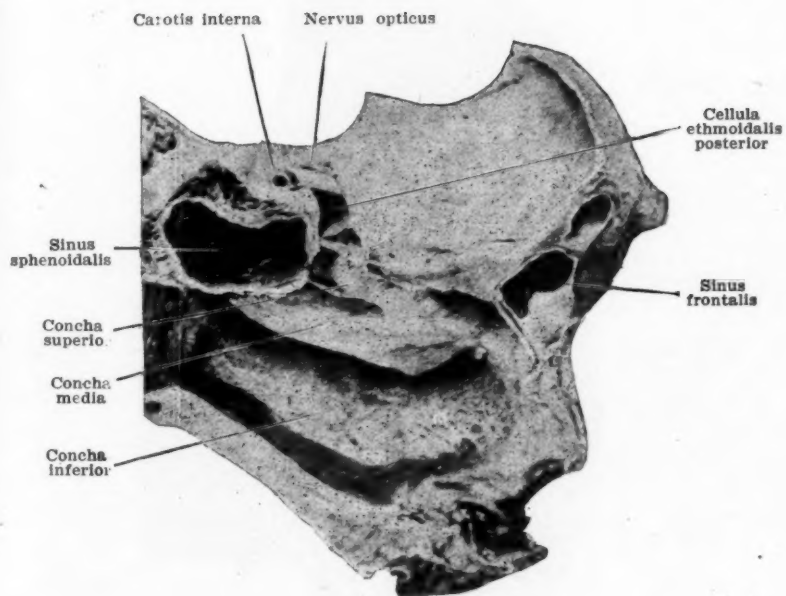


FIGURE 12.

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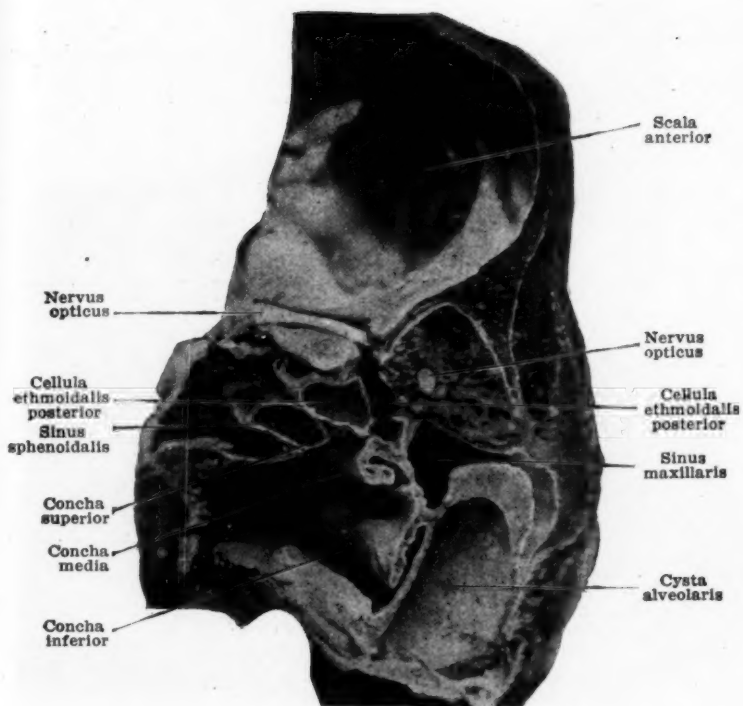


FIGURE 13.

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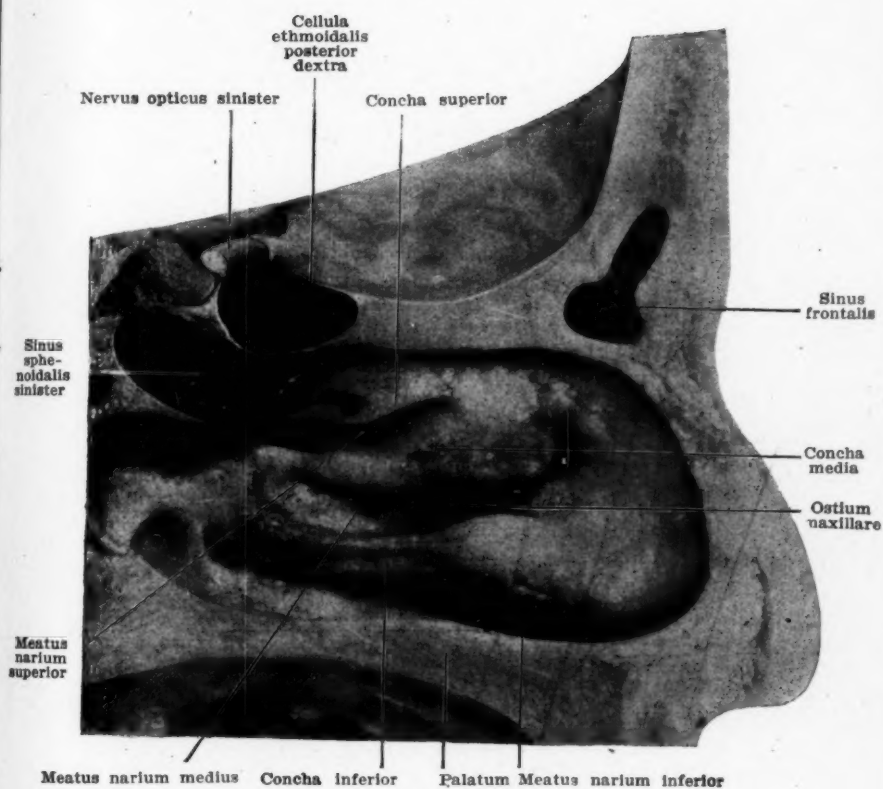


FIGURE 14.

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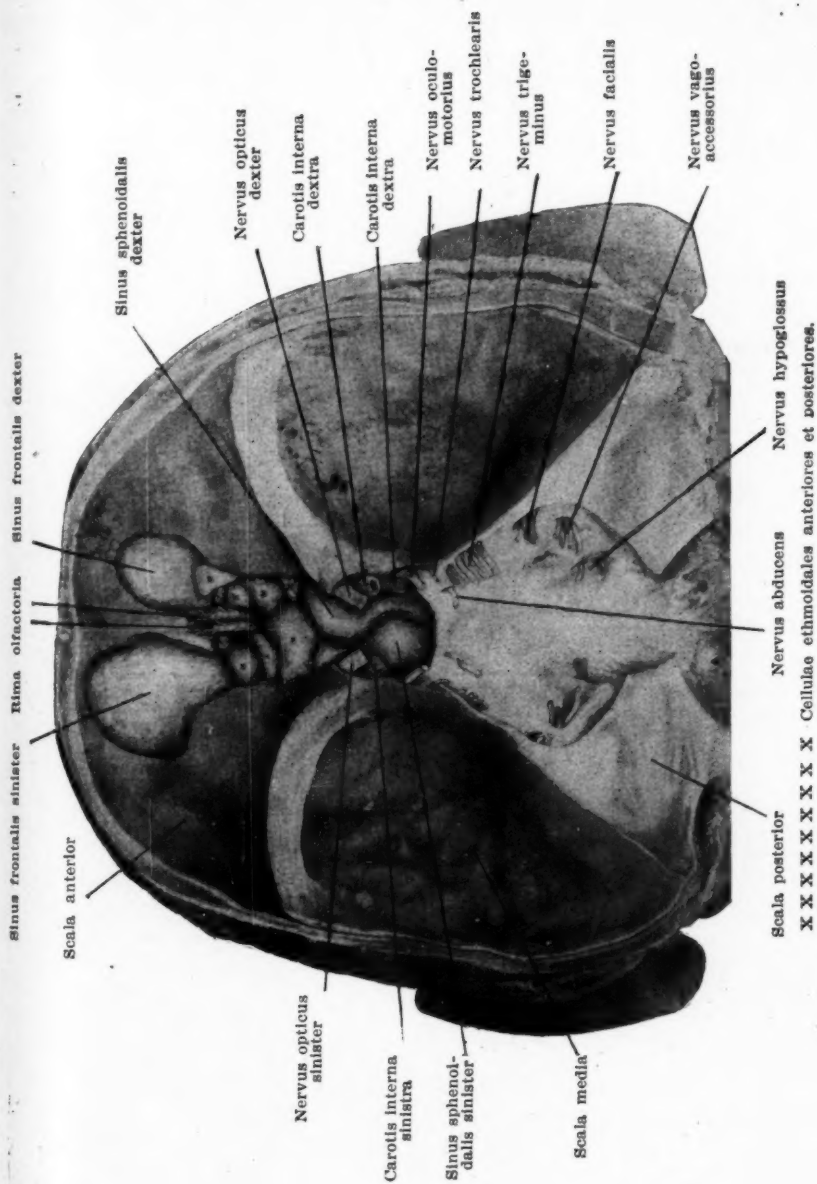


FIGURE 15.

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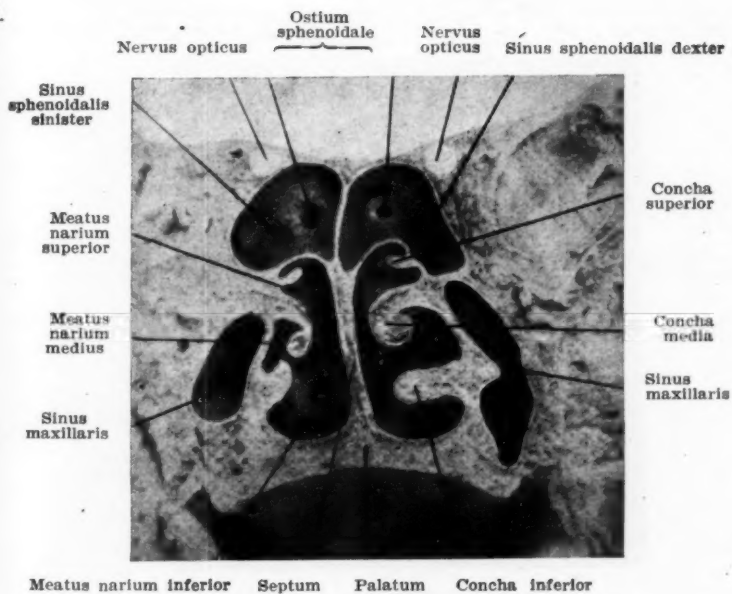


FIGURE 16.

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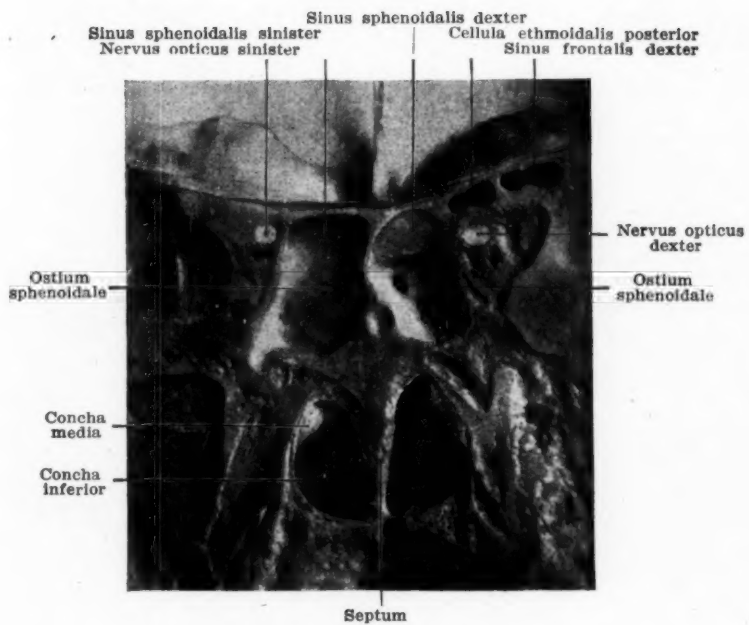


FIGURE 17.

THE
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Nervus opticus dexter Sinus sphenoidalis dexter Nervus opticus sinister

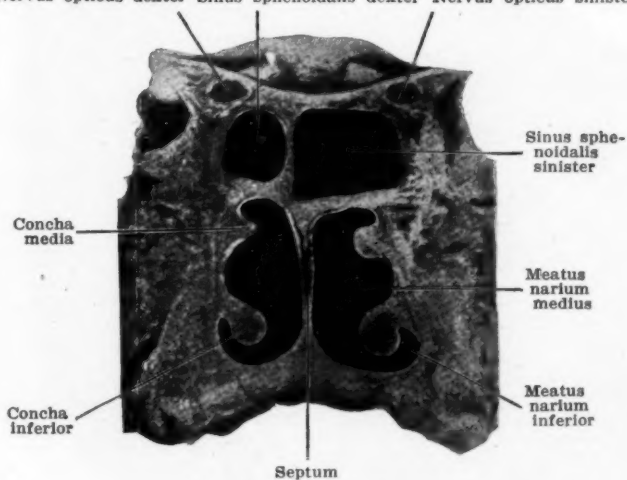


FIGURE 18.

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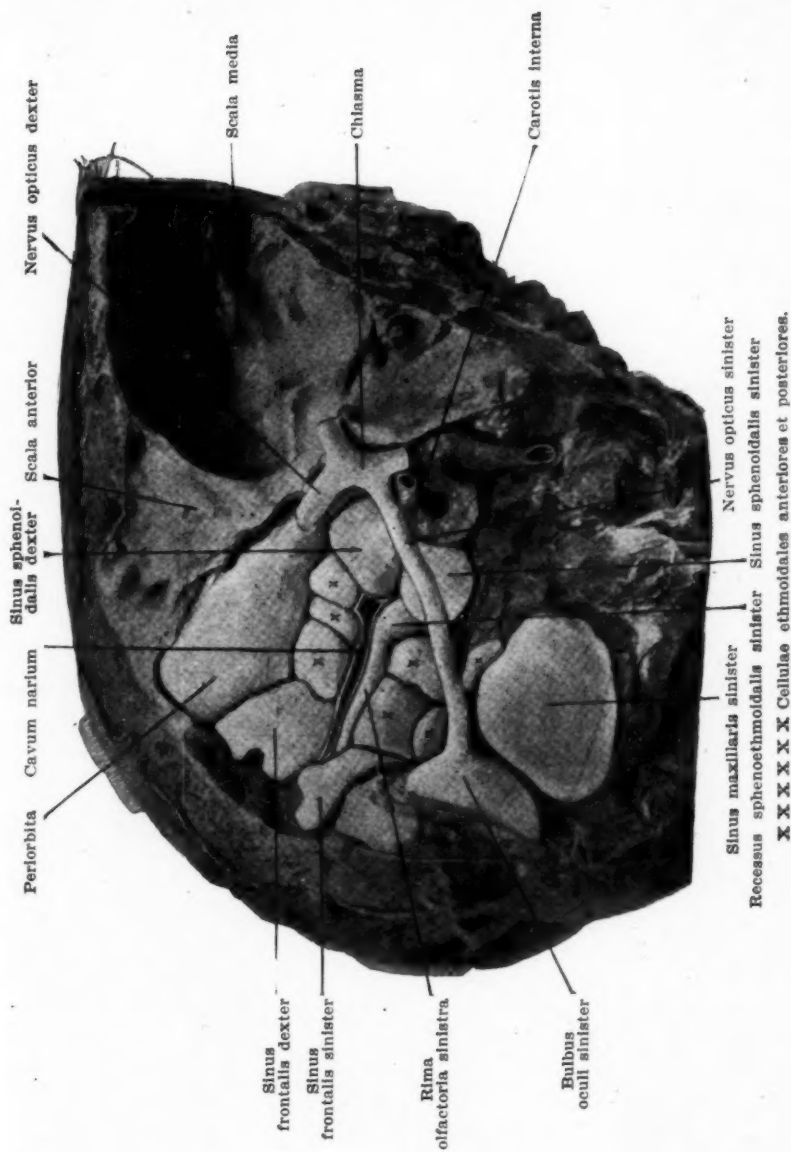
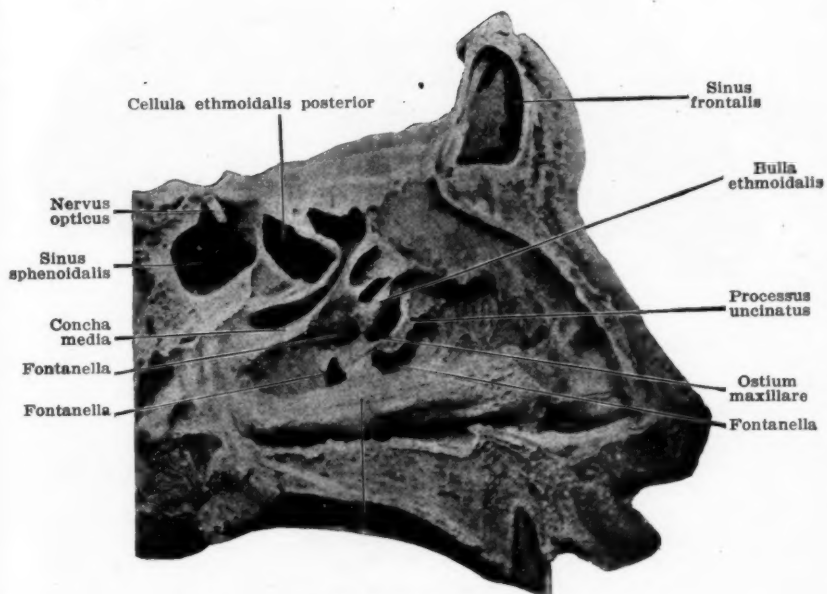


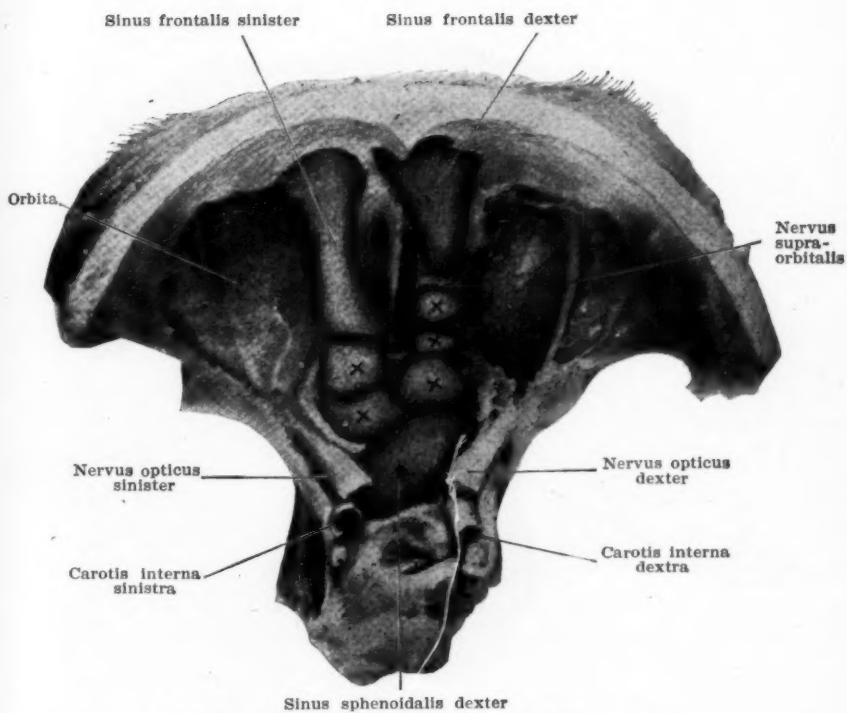
FIGURE 20.

THE
JOHN C. BERA
LIBRARY.



Concha inferior
FIGURE 21.

THE
JOHN C. C. C.
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X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 22.

THE
JOHN L. HERMAN
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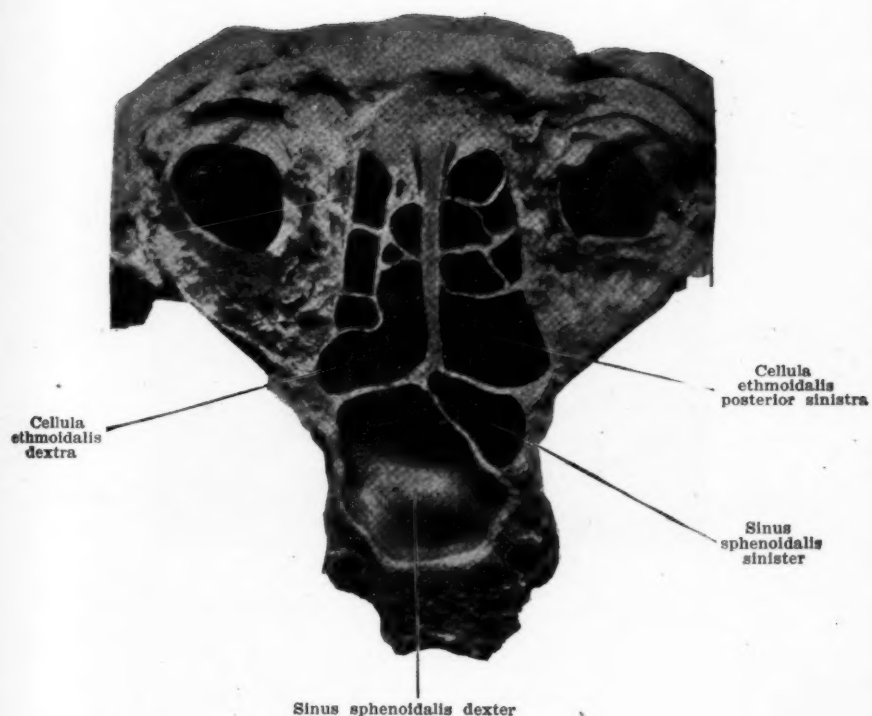


FIGURE 23.

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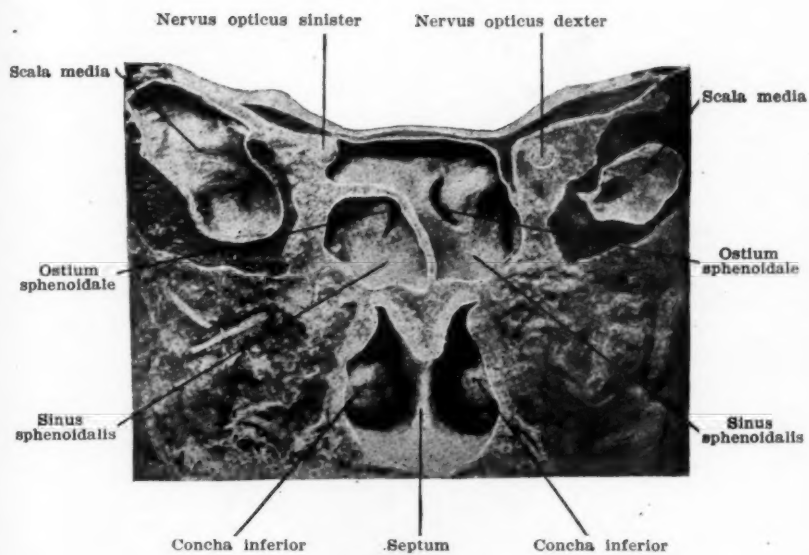


FIGURE 24.

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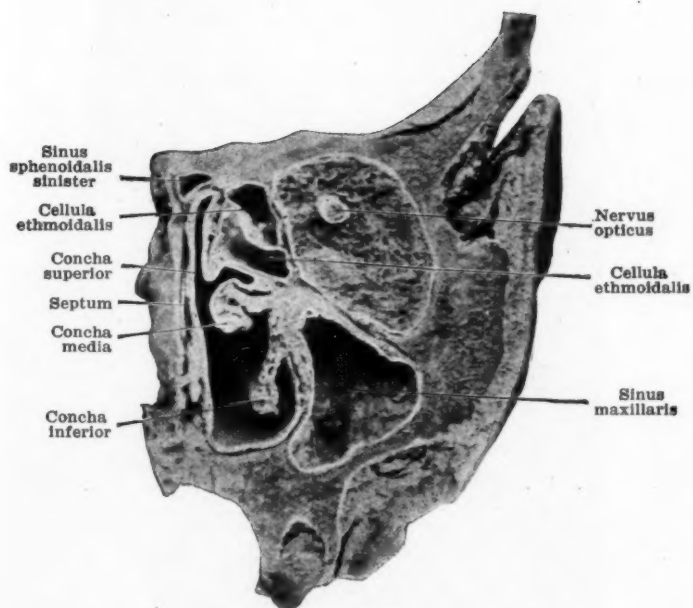
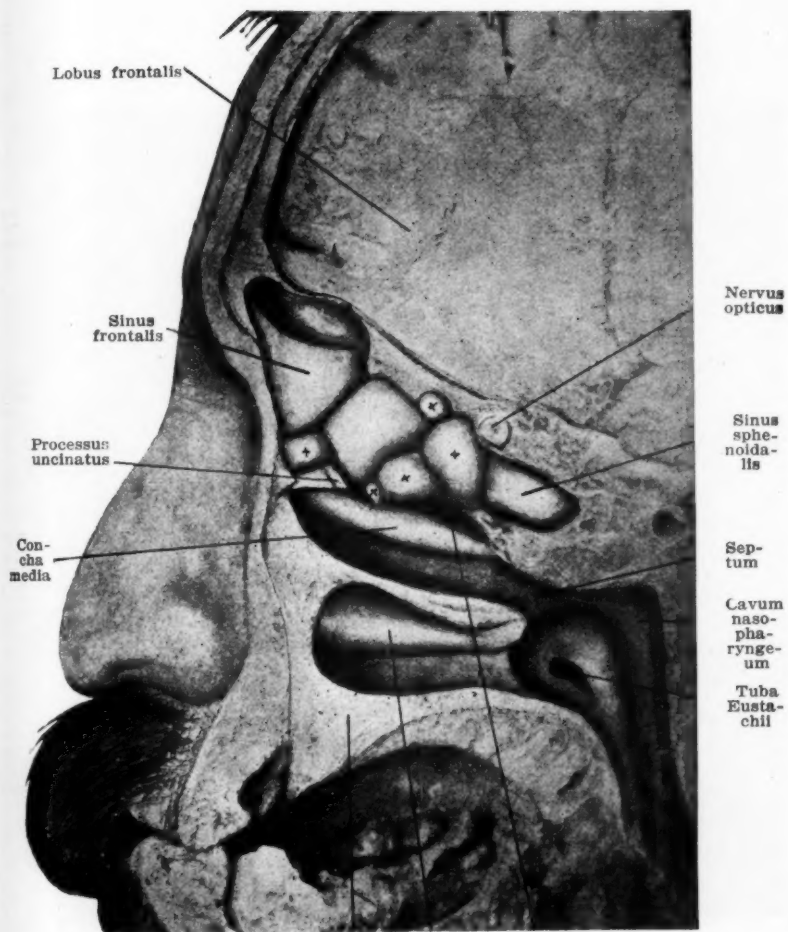


FIGURE 25.

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Pr
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Palatum Concha Meatus narium
inferior superior

X X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 26.

THE
JOHN CRERAP
LIBRARY.

Sinus
fron-
talis

Sacculus
laci-
malis

Bulbus

Sinus
maxil-
laris

Processus falciformis

Scala anterior

Sinus
fron-
talis

Sacculus
lacri-
malis

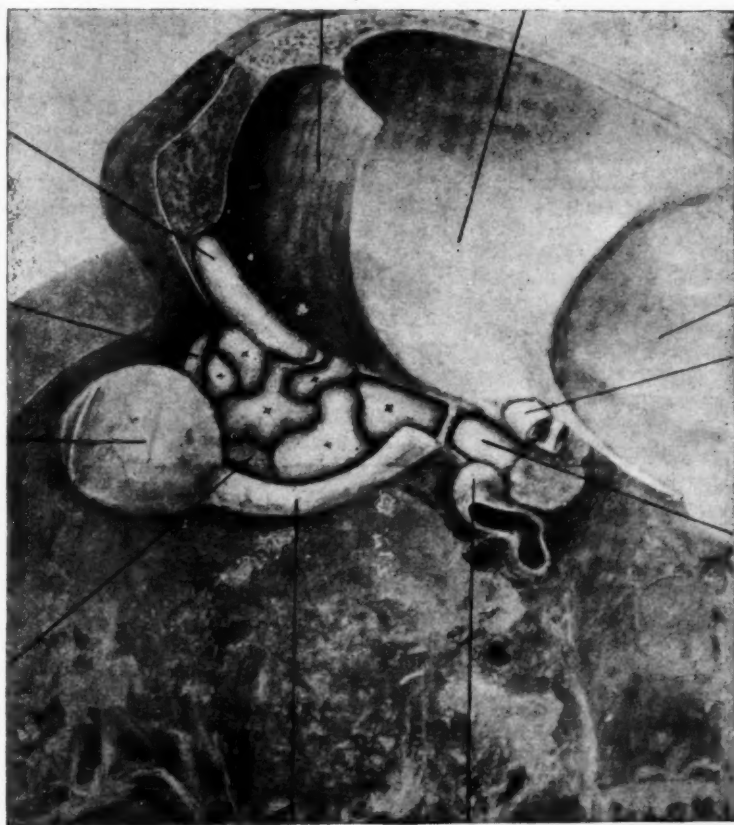
Bulbus

Sinus
maxil-
laris

Scala
media

Nervus
opticus
dexter

Sinus
sphe-
noidalis
sinister



Nervus opticus sinister

Carotis interna sinistra

X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 27.

THE
JOHN C. ORE
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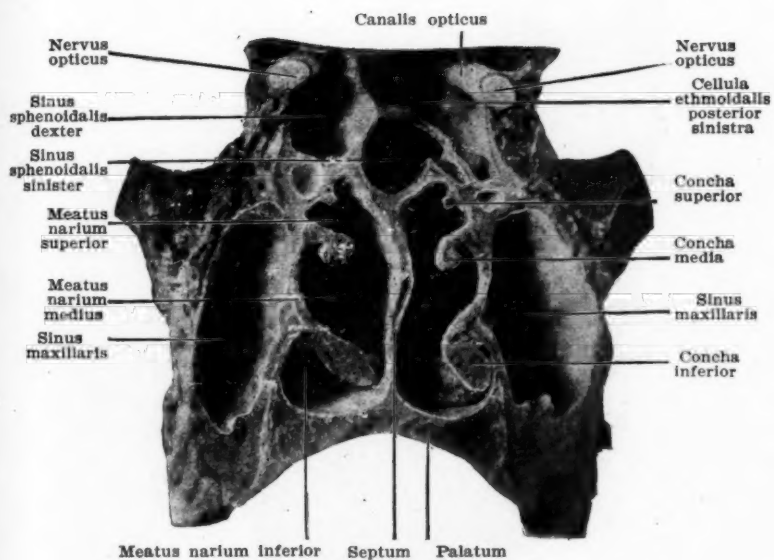


FIGURE 28.

THE
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Nervus opticus

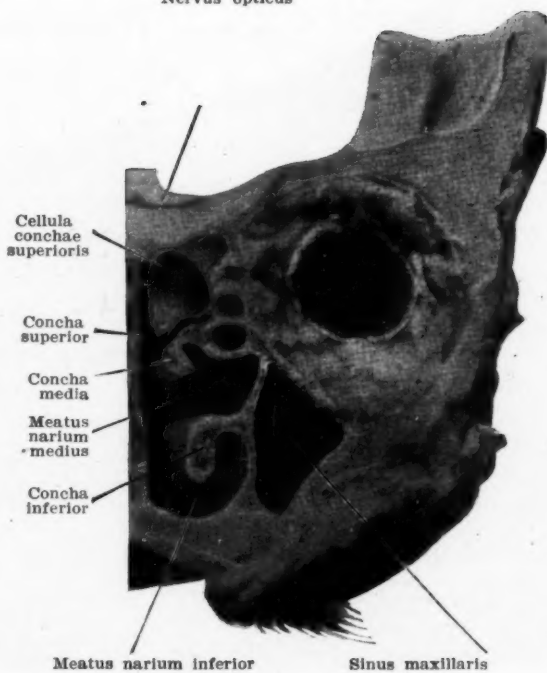


FIGURE 29.

THE
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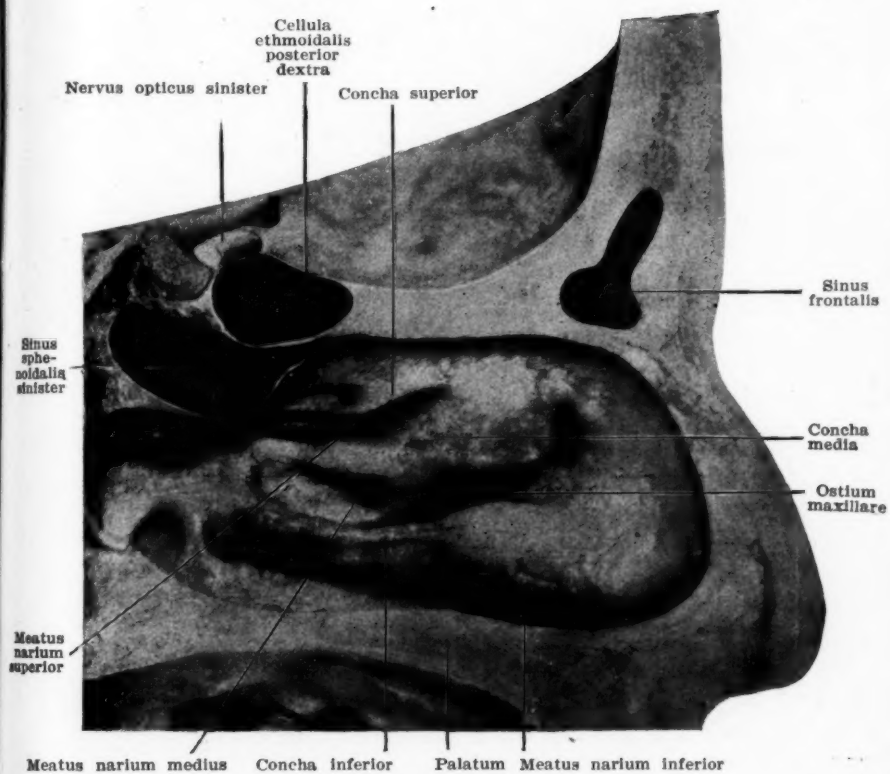


FIGURE 30.

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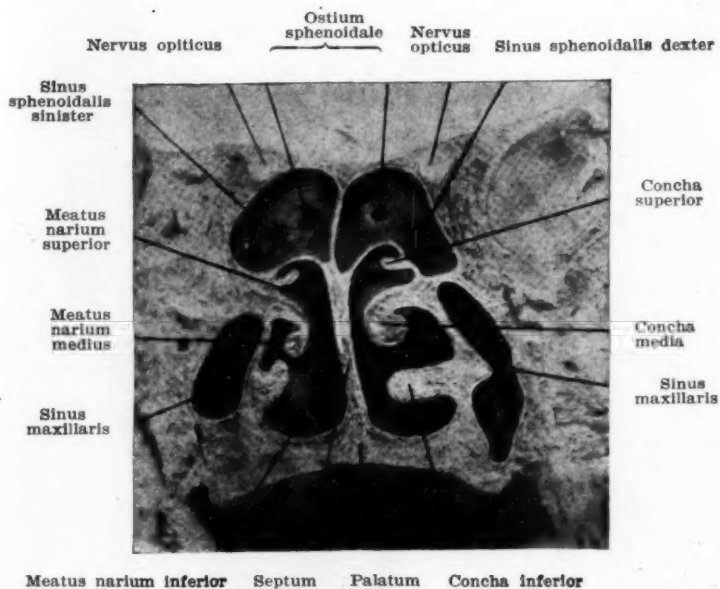


FIGURE 31.

THE
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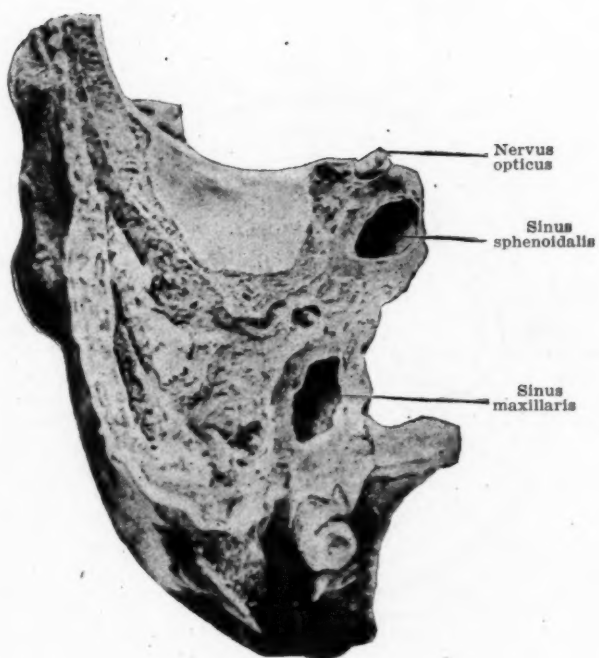


FIGURE 32.

THE
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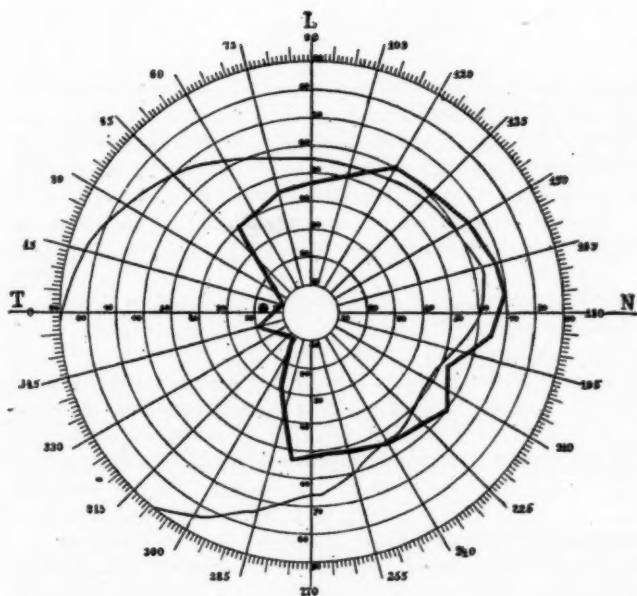


FIGURE 33.



II.

OBSERVATIONS ON TEN CASES OF INTRACRANIAL COMPLICATIONS OF MIDDLE EAR SUPPURATION.

By S. J. KOPETZKY, M. D.,

NEW YORK.

The following cases came under observation during the last two years, and because of the variety of the intracranial lesion and the results obtained, are believed of interest. A brief outline of the case histories, covering the absolutely essential details only, has been deemed sufficient, space forbidding an exact repetition of the daily record for each individual case.

CASE 1.—F. M., aged 27; came under observation in August, 1906.

She had measles, scarlet fever and whooping cough in childhood. Following a cold three years ago, there developed a discharging ear. For a few days before her appearance at the Manhattan Eye and Ear Hospital, there appeared pain in the right ear and a swelling behind the auricle. Some tenderness upon pressure below the tip was evident.

Diagnosis.—Acute exacerbation of a chronic middle ear supuration.

Operation.—On August 20th, 1906.

Operative Findings.—Mastoid process completely eburnized. Antrum, when uncapped, found small in size, containing cheesy deposits. Tegmen antri et tympani opened, evacuating extradural abscess. Original opening through tegmen accidentally made. The pus of the abscess is of foul odor. The dura is freely exposed; it evidences pulsation and is covered with dirty granulations.

The operation completed, wound washed and packed; and external dressings applied.

Postoperative Period.—August 21st, patient comfortable. Pulse, 114; respiration, 24; temperature, 100.

August 22nd.—There was nausea and vomiting. Pulse, 124; respiration, 24; temperature, 100.3. The patient complains of

feeling dizzy and occasionally as if falling forward. No nystagmus present.

August 23rd.—Wound dressed. The dura appears necrotic, with pus oozing from between dura and bone. The general appearance of the patient seems good, although an increase of nervous irritability is noted. During the day, vomiting spells occurred three times. Pulse, 90; respiration, 22; temperature, 99.

August 25th.—Some horizontal nystagmus noted when eyes are turned toward the healthy side. Later in the day, in addition, vertical nystagmus is apparent. Pulse, 90; respiration, 22; temperature, 100.

Exploratory operation was decided upon because of the persistence of purulent discharge from between bone and dura and because of the apparent breaking down of the exposed dural area, in addition to the continuance of the system's symptoms and nystagmus.

Explorative Operation.—The tegmen was further removed, working medially toward the pyramidal tip and semicircular canals. A small fistulous opening was now revealed in the dura, which, upon further exploration, was found to communicate with an abscess cavity. The cavity was wiped clean and a drain placed in position. Packing of the mastoid wound and external dressings followed.

The patient gradually grew worse, the septic appearance became marked until on August 28th, typical septic temperature was established. Patient was semiconscious and choked discs were present. The dura was examined under the squamosal plate, and the brain cortex found necrotic in areas. This was scraped away. Pus was evident near the tip of petrosal pyramid. These procedures were made under anesthetic. Exploratory puncture failed to locate deep seated abscesses. Death three and one-half hours after exploratory operation.

CASE 2.—J. M., 1 year of age; came under observation March 27th, 1907. The left ear had been discharging for three months. The present illness began with vomiting and loss of weight for some time before the ear discharged.

Simultaneously with the appearance of the otorrhea, a facial paralysis became noticeable.

The patient is much emaciated, semiconscious. The drum membrane is destroyed, the middle ear cavity filled with granu-

lations. Radical mastoid operation performed at the Manhattan Eye, Ear and Throat Hospital, March 28th, 1907.

Operative Findings.—Exposure of cortex revealed a fistulous tract leading directly downward toward the antrum. A second fistulous tract was found spreading backward toward the sigmoid sinus and toward the dura. Both dura and sinus were uncovered and examined. The dura was blanketed with discolored granulations, and at one locality a distinct necrotic ulcerated area was found.

The posterior meatal wall was partly involved in the necrotic lesion, involving the facial nerve whose distal end was imbedded in granulations. The proximal end of the facial nerve was not found. The horizontal semicircular canal wall was necrosed and the canal's lumen open.

Postoperative Period.—At the change of dressing April 1st, 1907, the bandage and dressings were noticeably fouled. The patient was fretful and screamed when moved.

April 2nd.—Muscular spasms of slight nature noted. The child refuses nourishment.

April 3rd.—Dressed. Foul odor in dressings.

April 4th.—Vomiting. Muscular twitching continued. Nystagmus appeared.

April 5th.—Foul odor to dressings continue. Granulations beginning to appear. Slight secretion in external auditory meatus.

April 6th.—Spasms almost continuous. Head and neck rigid and bent back. Nourishment not retained. Fed by rectum, but not retained. Temperature, 100; respiration, 48; pulse, 158.

April 7th.—Temperature, 101; respiration, 38; pulse, 136. Vomiting constantly. Child unconscious.

April 8th.—Temperature, 101.2; respiration, 40; pulse, 156. Jaws locked.

April 9th.—Temperature, 103.6; respiration, 36. General condition unchanged.

April 10th.—Temperature, 104.6; respiration, 24; pulse, 90. Child alternately rigid and relaxed. Nourishment partly retained. Dressing of wound showed dirty. Toward end of day temperature dropped to 102 and again rose to 103.6.

April 11th.—Patient rapidly sinking. Temperature, 104.4. dropping as death comes on.

No second operation was undertaken in this case because of the absolutely poor general condition after the first operation.

The child was brought in semiconscious and never rallied. The radical operation at that time included exposure and exploration of the meninges.

CASE 3.—N. P., aged 1½ years, appeared at the ear clinic of the Manhattan Eye, Ear and Throat Hospital, April 16th, 1907.

Patient had been ill for over a year with gastro-intestinal disturbances. The child was breast fed. For one year there had been discharge from the left ear. For about a week the ear appeared to give pain when touched. For three days the child has had fever. The ear evidenced a profuse otorrhea, with foul odor, the drum presented a large defect through which profuse granulations sprung. The discharge showed a mixed infection. Status on admission, temperature, 101.4; respiration, 56; pulse, 134.

The case was kept under observation for forty-eight hours. On the day following admission, April 17th, the temperature rose to 104, then dropped to 98, rising again to 102.4. Radical operation was performed on April 18th, 1907.

Operative Findings.—Upon retraction of the soft parts, a perforation was found through mastoid cortex, just below the level of antrum. The mastoid cells were filled with pus, granulations and detritus. The tip was removed. Inner table of mastoid was outlined. Postmeatal wall was lowered, connecting the tympanic cavity with the antral opening. Low wall of the meatus was found necrotic and was partially removed. Eustachian tube curetted. The horizontal semicircular canal wall was found eroded. Wound cleansed, packed and dressed.

Postoperative Period.—Following the radical operation, the child did well, from April 19th to April 23rd. No abnormal temperature rise noted. On April 23rd, Panse flap was cut, placed in position, postauricular wound sutured and the dressings reapplied.

April 24th.—Beginning facial paralysis becomes evident. The temperature rose late in the day to 100.6; respiration, 52. pulse, 150. A rash appeared covering the entire body. Thinking it perhaps scarlatina (for it looked so), a diagnostician from the health department was called in, who declared the rash non-contagious. Some cough developed and on April 24th, the temperature rose to 103.8; pulse, 159, respiration, 50.

Two small areas of consolidation became evident, in the lungs, in the postscapular region.

On April 25th, the rash is still present. The case was seen by attending pediatrician and pronounced non-contagious. The rash appeared like small petechial spots.

Dressing changes meanwhile showed mastoid wound to look healthy. Facial paralysis still present.

April 26th.—Temperature fell, gradually reaching normal. The pulse rate and respiration dropped proportionately. The rash is still present on face and body.

The child improved and was finally discharged from the wards and sent to the out-patient department. May 2nd, still under personal observation. Facial paralysis complete.

From May 2nd to 11th I saw and dressed the wound in the out-patient department.

May 11th.—Child was brought in with temperature of 103; pulse, 128; respiration, 28. Child refused nourishment, is restless and semiconscious, and head slightly drawn back. Smear from wound gave streptococcus infection. Immediate exploratory operation.

Operative Findings.—The old mastoid wound cleaned out. Incision lengthened upward. Periosteum retracted. Squamosal plate opened, dura exposed. No pus found. Dura opened. Brain found normal.

Posterior and superior semicircular canals opened. The promontory opened and the vestibule and cochlear whorl cleansed. Pus was found in the internal ear channels. The stapes was imbedded in a mass of granulations. During the manipulation about the stapes nystagmus was absent.

Skull cortex behind the mastoid wound was opened, dura over cerebellum discolored. Incision released discolored and purulent cerebrospinal fluid under pressure.

A lumbar puncture made at the time of operation gave only 3 cc. of cloudy fluid. This fluid showed 71 per cent polymuclear leucocytes, and 29 per cent of lymphocytes. Smears from the puncture fluid gave a small number of streptococcus chains.

The child's condition was bad immediately after operation and suitable stimulant restoratives were administered.

May 12th.—Temperature ranged from 103 to 104; the pulse from 150 to 160 and respiration from 44 to 78. Generally, the condition was unchanged.

May 13th.—A second lumbar puncture was made. The fluid was thick and tinted. Not found under pressure and only about 8 cc. withdrawn.

May 15th.—Lumbar puncture obtained only two drops of a very thick fluid. Temperature between 103 and 104; respiration, 54-60; pulse, 140-168. The general condition worse. Death, May 18, 1907. Temperature, 103; respiration, 98; pulse, 180.

After each puncture, and especially after the escape of fluid upon incision of the cerebellar dura, the rigidity of the head and neck disappeared, and the child appeared brighter. The failure to obtain fluid at puncture under pressure, when it was shown in the subarachnoid space under high tension, is accounted for by a closure of the foramen of Magendie.

CASE 4.—F. T., aged 15 months, came under personal observation, giving the following history:

For about a week the child suffered from gastro-intestinal disturbances and loss of weight. Child had had fever. Both ears were suspected because of evident distress when they were handled. Paracentesis was performed on both ears, June 10, after which otorrhea became established. Mastoid process was tender to touch and some swelling was noticeable behind the ears.

Expectant treatment was continued till June 13th, when child was admitted to the hospital, with temperature, 102; respiration, 20; pulse, 140, and simple mastoidectomies were performed on both sides.

Operative Findings.—Retraction of soft parts showed an intact cortex. Antri opened and filled with pus. Mastoid cells eviscerated. No sinus or dura was exposed.

The wound cavities were irrigated with normal salt solution, permitted to fill with blood and subsequently treated by the blood clot aftertreatment, subcutaneous sutures being used.

The temperature dropped to normal upon the second day after operation and remained normal thereafter.

June 16th.—A slight odor noticeable in the dressings from the right ear, some serous oozing from the lower wound end. The odor continued to be noticed, and finally on June 17, 1907, also appearing on the left side, the wounds were opened, the cavities washed with antiseptics and subsequently dressed with the usual open method.

Thereafter the case continued uneventful, the granulations gradually filling in the cavity, and the wounds on both sides becoming fully healed.

July 10th.—Both ears being examined and found dry, the case was discharged. Control examination, July 20th.

For about one month nothing was seen of the patient.

August 10th.—The child awoke during the night, as reported by the mother, with a facial paralysis on the right side. The child vomited during the night a number of times, and the following morning was readmitted to the hospital. Temperature, 103; respiration, 28; pulse, 130; facial paralysis and otorrhea on right side.

That afternoon muscular twitchings noted in hands and arms. Pupils dilated. Head drawn backward. Some convulsions immediately before operation. Temperature at time of operation, 103; respiration, 50; pulse, 130.

Exploratory Operation.—August 12, 1907.

Operative Findings.—Incision along old scar on right side. Periosteum retracted, and radical mastoid operation performed. Necrotic bone discovered in antral region, and over inner tablet of mastoid covering middle cranial fossa. The postmeatal wall lowered. Tympanic cavity cleansed, ossicles removed. Anterior mastoid plate lowered. The stapes found imbedded in granulations, with perforation of its annular ligament. The labyrinthine windows were opened and connected, cochlea opened, and horizontal semicircular canal opened. The vestibule was cleansed. Facial nerve was exposed. Dura was exposed and found healthy in appearance. Pressure on jugulars caused increased flow from opening in semicircular canal.

Following the operation, more convulsions noted. All limbs involved. At midnight, vomiting and extreme restlessness.

The following morning temperature, 104.2; respiration, 36; pulse, 140. Later respiration spasmodic, pulse weak.

A difference in the size of the pupils noted. Right pupil larger than left. Death soon followed.

CASE 5.—A. R., aged 18 years, was admitted May 11, 1907, giving the following history. Had typhoid-malaria several years ago. Never had any of the exanthemata.

Left ear has been discharging on and off since infancy whenever patient caught a "cold."

Her present illness began three weeks ago, with pain in the ear, and discharge.

Examination showed absence of edema about pinna. Marked pain on pressure over tip and lower portion of mas-

toid generally. Otosopic examination revealed perforation in lower posterior quadrant, discharging thin purulent matter. No sagging of canal walls, no headache, twitching of muscles or vomiting. General appearance septic. On admission, temperature, 100; respiration, 20; pulse, 92. Immediate operation was deferred because patient seemed comfortable and urine showed marked albuminuria with red and white cells, granular and hyaline, and epithelial casts.

Ear smear showed a few extracellular diplococci.

Patient was put upon a milk diet, diuretic administered and the ear locally treated.

May 12th.—Morning temperature showed 100.4; respiration, 24; pulse, 96. In the afternoon patient had a chill and temperature rose to 105.4; respiration, 22; pulse, 126. A foul smelling otorrhea followed paracentesis. Immediate operation was ordered.

Operative Findings.—Mastoid found eburnated. Cortex opened and pus located in a tract leading from tympanic cavity to the sigmoid sinus whose walls were found opened and whose lumen contained fluid pus. No bleeding obtained from the sinus. The inner tablet was removed backward, to within one-half inch of the torcular before blood was found. From bulbar end no blood was obtained.

Without curetting bulb and avoiding all unnecessary manipulations, the internal jugular vein was laid free, tied off about one inch above the clavicle and resected to the point of entrance of the facial vein. Then the jugular bulb was curetted.

The wounds were cleansed, packed and dressed. Purse string sutures were used on the neck wound.

Postoperative Period.—Following operation, the temperature promptly fell to 100.4; respiration, 26; pulse, 124.

May 13th the urinalysis showed albumin negative, with absence of red, white and epithelial cells. A few hyaline, granular casts were still present.

May 14th.—Temperature, 98.4; respiration, 24; pulse, 98. Patient generally comfortable.

Dressing changes showed lower wound perfectly clean. String sutures drawn together and lower part of wound closed. Mastoid wound clean. Plug changed over torcular end of sinus. From May 14th to May 19th, temperature ranged from normal to 99.8, lower in the morning than in the

evening. The pulse ranged from 88 to 96. The respiration from 20 to 22.

In general, conditions seemed so favorable that the patient was permitted to get out of bed.

May 21st.—Headache was complained of.

May 20th to 22nd.—Temperature about 100; pulse 80 to 90; respiration, 20 to 22. The dressings changed May 22nd gave an offensive odor from upper wound. Appearance healthy, however. Lower wound (neck) clean, middle ear somewhat dirty looking.

Patient was put to bed because of occipital headache, some nausea and vomiting. Temperature, 100.6 to 102.6; respiration 22 to 24; pulse, 96 to 110.

May 23rd.—Eyes examined. Slight blurring of discs on temporal side. Some lateral nystagmus.

No dizziness, patient stands quietly and does not stagger when walking.

May 24th.—Lowest temperature, 99.6; highest, 101.4; respiration, 20; pulse, 96 to 106. Patient appears to be drowsy.

May 25th.—Dressed. Very foul odor present in posterior part of mastoid wound. Some pus is evident at upper part of neck wound. Temperature ranged between 101 to 101.4; pulse, 100 to 104; respiration, 22.

After consultation with Dr. Wendell C. Phillips, exploratory operation was undertaken.

Exploratory Operation.—Posterior wound extended backward. Cortex further removed. Visceral side of sinus wall examined and found healthy. Dura over cerebellum examined and found healthy. Bulb examined and found free from pus. Irrigation of wound, and packing replaced.

May 26th.—Temperature, 101.4 to 100; respiration, 20 to 22; pulse, 98 to 90. Patient vomiting frequently.

May 27th.—No change.

May 28th.—Patient very drowsy. Occipital headache. Gradually became weaker, and comatose.

May 29th.—Vertical nystagmus noted. Left pupil larger than right. Right fundus and disc blurred.

May 30th.—Death.

Postmortem examination of mastoid wound gave pus behind visceral wall of sigmoid sinus, extending toward cerebellum; extent not determinable as autopsy was refused.

CASE 6.—(Seen in association with Dr. R. Johnson Held).

Male, aged 52 years, had suffered from chronic otorrhea for three years. For six months previously had indefinite pains over mastoid region. Three weeks ago facial paralysis came on.

Examination showed all symptoms of mastoiditis on the right side, with facial paralysis. Temperature, 101; pulse, 92; respiration, 22. The patient seems indifferent to his condition, exhibiting retarded cerebration. There was marked tenderness over entire mastoid. Considerable thick discharge from the ear, greenish in color and of a foul odor. Tympanic cavity was full of granulations, making the membrana tympani invisible.

Radical operation performed April 24, 1906.

Operative Findings.—Upon removal of cortex, considerable pus, granulations and bone detritus were uncovered. The entire cellular character of the mastoid had been destroyed under the purulent attack. The internal mastoid table was absent, exposing the sinus and cerebellar dura. These were covered with discolored granulations. The tegmen aditus ad antrum was absent. The postmeatal wall was destroyed, including the major part of the external (lateral) aditus wall. In other words, "Nature had attempted the radical."

After cleansing, examination revealed absence of the tympanic floor, the jugular dome being free in the contents of the tympanic cavity. The facial nerve was found exposed, diseased and imbedded in granulations. The bony framework of the Eustachian tube had disappeared, as well as the anterior tympanic wall, exposing the capsule of the joint of the lower jaw at the glenoid fossa.

The dura over aditus and tympanic cavity was found perforated, pus exuding. Upon exposure of the brain substance, a sloughing ulcer of the brain surface was uncovered, 1.5 cm. in area, 0.5 cm. in depth.

The wound was cleansed, packed and dressed. The patient made a complete recovery. Facial paralysis persisted. Upon inquiry, I learned that he died of some intercurrent disease one year later.

During the entire postoperative period the temperature never rose above 100.

CASE 7.—(Seen in association with Dr. R. Johnson Held and reported by us in the *Archives of Otolaryngology*, Vol. XXXV, No. 6, 1906; republished here to complete histories of mate-

rial from which paper was written). R. R., aged 3 years, appeared at the Manhattan Eye, Ear and Throat Hospital, December 13, 1904, suffering with a chronic middle ear suppuration. The radical operation was performed April 29, 1905. At this operation, necrosis of bone was found, extending up to the dura, over the tegmen. The left ear was operated upon March 13, 1906, the radical mastoid operation being performed. In this operation the dura was exposed because bone necrosis extended to and involved the tegmen. The dura was found covered with dirty granulations.

On May 8th symptoms of meningitis came on. The head was markedly retracted, the pupils dilated, the right side deviated outward. The patient was restless and cried continuously. Temperature, 102.4; pulse, 144; respiration, 75. Examination revealed pus in the wound cavity.

Exploratory operation was performed May 12, 1906, the patient having been in a condition of stupor and unconsciousness for twenty-four hours.

Operation.—The old mastoid wound was reopened and cleansed, all granulations were removed, the dural surface exposed and discolored granulations upon it cleaned away.

The lower part of the squamosal plate of the temporal bone was opened and a considerable extent of its area ($3 \times 3\frac{1}{2}$ cm.) removed. Through this, the dark congested dura bulged. The dura was incised by horizontal incision, extending the entire length of the exposure. From this incision purulent fluid escaped. Brain substance immediately protruded into the wound. The brain tissue was extremely friable. The wound was flushed with hot saline solution and the entire surface carefully wiped. The brain was carefully lifted, and wiped underneath, and a strip of gauze introduced between brain and dura downward and inward toward the interior of the skull. The gauze was kept in one long strip, the end of which protruded into the external dressings. The mastoid wound was then flushed, packed and dressed. Temperature chart on the next day showed: Temperature, 105; pulse, 170; respiration, 80.

May 14th—The left lateral ventricle was punctured and one and a half drams of purulent fluid withdrawn. Lumbar puncture showed 40 cc. of grayish-white turbid fluid. Following these measures, an improvement was noted. Temperature ranged from 101 to 103.2; respiration, 24 to 30; pulse, 120 to 140.

May 16th.—Lumbar puncture was again performed and 30 cc. of turbid fluid withdrawn. Temperature chart gives temperature, 99 to 101.8; respiration, 26 to 30; pulse, 100 to 140. There is less restlessness, and patient had natural sleep.

May 18th.—Lumbar puncture gave 35 cc. of clear limpid fluid. This had some white sediment, some blood, but no coagula.

Decided improvement in the general condition. Less retraction of head, less restlessness, etc.

The first dressing was made on May 23rd. The wound appeared healthy and was covered with clean, healthy granulations. The dura still appeared thickened but of lighter color and only slightly congested. Temperature, 100; pulse, 106 to 130; respiration, 24 to 30.

From this on improvement continued. The intelligence cleared, temperature became normal, the rigidity of the back disappeared, the eye fundus clear, and case was finally discharged cured.*

CASE 8.—A. K., aged 16 months, came under observation October, 1907. The child had been sick about two weeks, with indefinite symptoms, some fever, gastro-intestinal disturbances, etc. For the last few days a swelling had been apparent behind the left ear. The swelling presents the appearance of subperiosteal abscess. The tympanic membrane was not perforated, and extremely bulging.

On October 27, 1907, simple mastoidectomy was performed.

Operative Findings.—Swelling over the cortex upon incision failed to evacuate pus. The tissues were indurated. The cortex upon retraction of the soft parts showed a perforation, the fistulous tract leading directly downward toward the antrum. Fluid pus was evacuated, coming away in pulsations. As the mastoid cortex was removed backward, the sinus wall was uncovered, liberating an accumulation of pus between its dural walls and the overlying bone. The sinus was coated with granulations. The tegmen was removed to evacuate pus extradurally.

The case made an uneventful recovery, until the fourth week of the after-treatment, when fever reappeared. The granulations were curetted and cleaned, and following this the case recovered.

*The merest mention of the principal findings of this case are here given, as a full and detailed report has already been published, as stated above

CASE 9.—A. H. E., aged 38 years, came under observation September 4, 1907. Following a cold, pain appeared in the right ear. Paracentesis was performed, and under local treatment by his physician, he apparently recovered, a discharge from his ear persisting, however.

After an interval of a week, the pain reappeared and the patient then came under my observation.

The right ear evidenced a small perforation, rather high up, from which pus came away under pressure. Paracentesis was repeated. Mastoid tenderness was evident only over the tip. There was no rise in temperature, and the patient seemed comfortable.

The pain gradually became more marked, and a swelling appeared over the mastoid, especially marked at the tip. This gradually spread upward, the edematous swelling finally reaching the upper pole of the auricular attachment.

On the drum, no marked bulging was evident, nor was there any sinking of the posterior superior wall.

Simple mastoidectomy was decided upon and the operation was performed September 25, 1907.

Operative Findings.—The incision into the postauricular swelling failed to evacuate fluid pus. The tissue was extremely edematous and indurated.

The mastoid was found to be broad from before backwards and unusually low from above downwards. Its cortex was very irregular, and the posterior root of the zygoma was very marked, stretching backward as a hard ridge of bone. The petromastoid was unusually well marked for an adult.

The retracted cortex evidenced a perforation, $\frac{3}{4} \times \frac{1}{2}$ inch, situated 1 inch above the broad flat mastoid tip. A probe introduced into this could be passed upward into the antrum and passed backward, pressing against soft granulation on the exposed sinus. The interior was filled with a blood-tinged pus.

The removal of the cortex, showed the entire mastoid process to have been disintegrated and filled with granulations and detritus. The mastoid process was only partly pneumatic. The tip was removed, the posterior meatal wall was found undermined by the purulent process and the facial ridge slightly involved. Of the posterior wall, as much as possible was removed, leaving only a thin ridge to support the external auditory canal.

The zygomatic cells were eviscerated, and the cells in the angle between the tegmen above and the sinus knee eviscerated.

The exposed area of the sinus was examined, cleansed of all pus, the granulations being left undisturbed. Further exposure of the sinus wall was made until healthy tissue was uncovered.

This case made an uneventful recovery.

CASE 10.—J. C., aged 22 years, came under observation October 10, 1907, giving a history of having had a cold, followed by pains in the right ear, four weeks previously. The ear has discharged profusely ever since. The patient has had continuous pain in the ear, and pain on pressure, with a fever for most of this time. Operative treatment had been repeatedly advised and had been persistently refused. For two weeks there have been frequent dizzy spells with nausea and vomiting. Lately the pain has subsided, but the dizziness, nausea and vomiting continue, and the patient is feverish, unable to take food, and so weak that she cannot stand unsupported. The general appearance evidences profound sepsis. Temperature, 103.8; pulse, 132; respiration 30. The external ear is full of foul-smelling pus, making a view of the drum impossible.

The ear stands off from the head, giving a postauricular swelling the appearance of a subperiosteal abscess. The greatest amount of induration is noted near the upper pole of the auricular attachment. Immediate operation recommended.

Operative Findings.—The incision into the postauricular swelling failed to show any pus present. The soft parts were found edematous and friable.

Retraction of the soft parts demonstrated an intact cortex. The first opening into the cortex evidenced pus coming away under extreme pressure. The entire cortex removed. Fluid pus and detritus eviscerated. The evisceration of the mastoid contents followed. Pus was traced to the tip which, when removed, revealed an abscess formation in the neck tissues. The cells spread far backward. Breaks in contiguity in the inner mastoid table showed pus behind this. The inner table was removed, uncovering an extradural abscess over the cerebellar dura. This was evacuated. The sinus was exposed, and because of its apparently unhealthy condition, plugged off, opened and examined. No thrombus was found.

The patient being in so precarious a condition, that anes-

thetic had to be suspended to administer restoratives, further procedures were curtailed to the utmost essentials, and after drainage of the antrum was established, further operative measures were suspended.

The next day the patient's condition was so precarious that it was not deemed advisable to give further anesthetic. The third day, however, her general condition was such that we deemed it advisable to proceed. Further procedures were deemed necessary, in view of an increasing somnolence and stupor and a beginning rigidity of the neck.

The Exploratory Operation.—The tegmen antri et celulae was opened to liberate a large quantity of pus between dura and bone. The radical mastoid operation was then finished. The dura was incised to afford intrameningeal drainage.

The patient did not recover from the general profound sepsis, and during the next twenty-four hours died.*

The youngest of these ten patients was 12 months, the oldest 52 years of age. The sexes were represented by six females and four males. The process was located on the right side in six cases, and on the left side in four cases. The ear disease had existed from two to four weeks in the acute cases; from 18 months to 17 years, in the chronic cases, before coming under treatment.

The acute conditions notably concerned children. A certain interval was noted between the first operation and the onset of the intracranial lesion. In two of these cases, the ear complication developing under sudden facial paralysis and a high fever. Another case was characterized by gastro-intestinal disturbances, and the absence of a discharge from the ear, attention being finally directed to the affected ear by the onset of otorrhea, together with the manifestations of facial paralysis. While the internal ear was involved in the intermediate lesion in these three cases, this extension of the pathologic process could be averted in another case, also concerning a child.

All the external evidences of subperiosteal abscess were present in three cases, but no fluid pus escaped from the indurated and edematous tissues overlying the mastoid process.

*Since paper has been in preparation, another case has come under observation, a child 10 months of age, with serous meningitis following double mastoidectomy (acute case). Meninges were opened and drained,—patient is recovering. Full report of this case will be given later, as it presents special features.

after the skin had been incised. In four instances, the mastoid cortex was found perforated (three children; 1 adult, aged 38 years).

There was inequality of the pupils in certain cases, the larger pupil in two instances corresponding with the diseased side. One case of sinus thrombosis showed marked albuminuria, which disappeared after the evacuation of the clot.

Five times the middle cranial fossa was invaded; four times the posterior cranial fossa alone; twice, both the middle and posterior cranial fossa.

The pathologic process, in a general way, advanced along two avenues: (1) The direct route, illustrated by seven cases, in which the infection traveled by contiguity from the middle ear to the inner table covering blood vessels or tegmen dura, with subsequent involvement of the meninges. (2) The indirect route, illustrated by three cases, in which the infection of the middle ear did not reach the meninges and brain until after preliminary invasion of the labyrinthine channels.

PATHOLOGIC LESIONS IN THESE CASES.

Cases.

Pachymeningitis externa (extradural and perisinus abscess)	7
Pachymeningitis interna (intradural abscess, cerebral and cerebellar)	4
Cerebral abscess (temporosphenoidal lobe)	1
Cerebellar abscess	1
Sinus thrombosis (involving jugular bulb)	1
Ulcerations on the brain cortex	1
Purulent labyrinthitis	3
Pus in the lateral ventricle	1

COMMENTS ON THE LESIONS.

The most frequent intracranial lesions of otitic origin consist in purulent inflammation of the external surface of the meninges and the formation of extradural abscess. As a rule, the process is referable to bone necrosis involving the dura, or it may develop in connection with caries or cholesteatomatous degeneration of the temporal bone. In the majority of the cases, the disease can be traced to the dura. A collection of pus in the cerebellar region appears in shape of a perisinus abscess. Although generally located over the tegmen, when

the middle cranial fossa is invaded, the pus has been known to occur at the top of the petrosal pyramid.

Bony disintegration is not necessarily complete, in abscesses complicating suppurative middle ear disease. Not infrequently, there is partial eburnization of the bone, with purulent tracts burrowing through it. The dura at the site of the abscess presents discoloration, and may be covered with a layer of granulations. Superficial ulceration, with purulent infiltration, has been noted on the meningeal membrane, which in other cases appears thickened and raised up from the bone, while a thick stream of fetid pus oozes between the meninges and the bone.

These variable findings I am inclined to interpret as pathologic stages rather than as types; the time at which the disease is first noticed representing an essential factor in this connection. Whereas nothing but a thickened dura may be found in a timely operation, delay would in all probability have led to the exposure of an ulcerated membrane. The findings are moreover to a certain degree dependent upon the species and virulence of the invading microorganisms.

Extradural abscess, provided it is exposed in time, with thorough removal of the pus, is very amenable to treatment.

Intrameningeal, or Intradural Abscess (*pachymeningitis interna*) occurs as a circumscribed lesion, without general infection of the subdural spaces, and is the result of disintegration of the dura, with involvement of the subdural spaces by the pathologic process. A certain amount of infectious material the dura is able to cope with,—in a way similar to the peritoneum,—without giving rise to a generalized infection. The intradural spaces are protected by the short duration of the local process, and the diminished virulence of the infection.

Koerner (*Die eitrigen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter*) describes two types of intradural abscess: (1) Subdural abscess, accompanied by ulcerations on the brain cortex. (2) The formation of intradural abscess, with a somewhat diffuse involvement of the intradural spaces. The writer interprets these conditions as differences of degree rather than of kind, in the sense outlined above. Cases of the first class may remain latent for a certain length of time, without giving rise to symptoms of any sort (Observation 6). As a rule, the middle cranial fossa is involved.

Surgical interference is followed by prompt relief, as illustrated by Case 6. The presence of a similar abscess can hardly be recognized prior to the operation.

The second group of cases is characterized by the simultaneous existence of extradural abscess, sinus thrombosis, or actual brain abscess (Observation I).

The clinical picture is dependent upon the distribution of the lesion and its manifold complications. In the vast majority of the cases, the diagnosis is not rendered until the patient comes to operation. A recognition at this time of the true state of affairs, with emptying of the pus, means a decided and speedy change for the better. The patient's prospects are less favorable in the diffuse conditions, where the inflammation reaches for into the intradural spaces (Observations 1 and 10).

Certain mixed cases occur, which present the features of both extradural and intradural abscess, sometimes in combination with rather deep ulceration or necrotic areas of the brain surface proper (Observation 6). These cases represent a transition form between extradural and intradural disease, and cannot be correctly included under either heading.

Purulent Otitic Meningitis occurs as an involvement of the pia mater, and may appear as an independent but simultaneous process, together with sinus thrombosis or brain abscess. Again, it may be the sequel to any of these lesions. It was observed as the terminal lesion in cases 1, 2, 3 and 4. It is a relatively uncommon, remote result of middle ear suppuration in its uncomplicated form; brain abscess or sinus thrombosis being the more usual sequela. Sometimes superficial brain ulcerations are noted. Martin (quoted by Huguenin, Ziemssen's Handbuch, Vol. IV), Bürkner (Arch. f. Ohrenheilk., 19, Case 3), Bezold (Arch. f. Ohrenheilk., 21, Case 5) and other observers reported small surface abscesses. The ventricles may be invaded by the pus, as shown in a personal observation.

The point where the meninges are in contact with the diseased bone is the favorite site of the greatest pus accumulation, which is accordingly looked for at this demonstrable point of contact. (Held and Kopetzky, Arch. of Otolaryngology, Vol. XXXV. The case upon which this observation was based, is republished in this communication as Case 7). This area constitutes the starting point of the meningeal involvement.

In those cases where a labyrinthine infection is responsible for the intrameningeal purulency, the meninges are generally found to have become primarily affected in the cerebellar region, as illustrated by observations 2, 3 and 4.

Although the exposure of the dura in the cerebellar region, in Cases 3 and 4, plainly showed extreme intracranial tension, the lumbar puncture did not demonstrate cerebrospinal fluid under pressure in the spinal canal. A considerable quantity of purulent fluid escaped from the arachnoid space, when the dura was incised, after negative pressure had been found at the lumbar puncture.

The discussion of the diagnosis of purulent meningitis does not lie within the scope of this article. The same remark applies to a detailed consideration of cerebral and cerebellar abscess.

Brain Abscess is usually produced by a chronic middle ear suppuration of prolonged standing (a number of years: three in Case 1; eighteen, in Case 2). The majority of brain abscesses are the result of actual disease of the bone, rather than lesions of the mucous lining of the middle ear cavities. For this reason, especial importance attaches to the differential diagnosis between a chronic middle ear suppuration associated with bone disease, and one in which the mucosa alone is affected. Although necessarily delicate, this differentiation is nevertheless possible to a great extent by the study of the otoscopic picture, combined with bacteriologic and cytologic examination of the discharges from the ear.

Adhesions between the dura and the surface of the brain are frequently observed in brain abscess, which should accordingly be suspected whenever such a condition is found to exist.

TREATMENT.

The treatment of these intracranial lesions is governed by the local findings in individual cases. Each patient will have to be treated according to individual requirements, and under these circumstances, it is not possible to map out a general plan of treatment adapted to all cases. The management of these lesions is based upon the following principles:

- I. The radical extirpation of the primary pathologic foci in the temporal bone, by means of a careful surgical exploration of the bony wound cavity (deep general anesthesia being usually required), for the uncovering and removing of

neglected purulent mastoid cells; with examination of the exposed sinus wall and the tegmen dura. A wider opening of the middle ear spaces is indicated in cases with a suggestion of labyrinthine involvement; here the wall of the labyrinthine capsule must be explored for evidence of the route of invasion of the internal ear. After the infection of the labyrinth has been demonstrated, a further exploration of the internal ear is required in the presence of defects in the walls of the semicircular canals, or ulcerations of the labyrinthine windows, or perforations in the cochlear convolutions. The internal ear is to be explored upon the basis of positive findings only, since this interference represents a very grave surgical procedure, which moreover implies the destruction of hearing in the affected ear.

II. The evacuation of extradural, intradural and brain abscesses, with establishment of meningeal drainage. The condition of the exposed sinus wall is revealed by its appearance, coloration, touch, etc. A perisinus abscess is emptied by the mere exposure of the sinus wall. The uncovering of a small area of dura over the tegmen is harmless in itself, provided the dura is healthy. Its systematic performance in doubtful cases will frequently serve to reveal the presence of unsuspected extradural gatherings of pus. Intradural lesions may be suspected upon the evidence of the exposed dura (aspect, color, feel) in combination with existing intracranial tension. This suspicion is confirmed when the dura is found to be covered with discolored granulations, pus escaping between the meshes.

Large areas of necrosis in the dura are removed, while small necrotic spots are allowed to remain. An incision of the dura provides for the escape of intradural pus. Large collections are best managed by means of exposure through the squamosal plate, and application of a counter-opening, for the facilitation of meningeal drainage. Multiple parallel incisions in the dura have been found serviceable. The evacuation of an abscess in the sphenoidal lobe, or puncture of the lateral ventricle, if required, is easier of performance from the opening in the squamosal plate, than by way of the existing wound.

Exploration of the sinus, and eventual ligation of the internal jugular vein, may become necessary by the development of sinus thrombosis, as a sequel to the evacuation of a perisinus ab-

scess. Under these conditions, surgical interference is indicated at once after the diagnosis has been rendered. A cerebellar gathering of pus is suggested by a continual high temperature, with a healthy wound, after ligation of the internal jugular vein, and evacuation of the clot, in sinus thrombosis. This indicates the removal of the inner mastoid table overlying the cerebellar meninges. After the cerebellar abscess has been located, the pus may be reached either by way of the mastoid wound, or through the cortex, posteriorly; the procedure to be adopted depending upon the location of the abscess in a given case.

III. The maintenance of normal intracranial pressure, as far as feasible, through repeated lumbar punctures. The diminution of intracranial pressure, through the withdrawal of cerebro-spinal fluid, is of importance, apart from any diagnostic data this may furnish. Relief and amelioration were invariably shown in the writer's experience, after lumbar puncture, by a clearing of the consciousness, a diminished rigidity of the back, and as a rule an improvement of the patient's general condition. The progressively diminishing purulence of the puncture fluids obtained from the favorable cases, at different dates, is of auspicious augury and stimulates further efforts in the interests of the patient. Meanwhile, such an outcome is reserved for the cases with a patent foramen of Magendie. The prognosis is a gloomy one when this outlet is obstructed, with a negative pressure of fluid in the spinal canal, and high tension of fluid in the subarachnoid space intracranially.

RESULTS OF OPERATIVE INTERFERENCE.

Among the ten cases, there were four recoveries and six deaths. The fatal outcome was due in one case to septic pyemia, the operation coming too late to prevent the effects of the profound general sepsis. In a second patient, a child, death occurred presumably from shock, before the operative results could be determined. In the remaining fatal cases, the surgical intervention failed to arrest the progress of the disease.

The fatal outcome is not necessarily governed by the wide distribution of the pathologic process. Recovery followed in one case of the series (Observation 7), although there was extradural and intradural pus, with involvement of the

lateral ventricle and spinal cord. A fatal ending was noted in those cases in which the posterior cranial fossa was the seat of the main foci of the lesion. The bone disease could be traced directly to the meninges by contiguity in only one of the fatal cases (Observation 10). The invasion of the posterior cranial fossa was shown beyond a doubt to take place by way of the labyrinthine channels, as illustrated by the gross operative findings in Cases 2, 3 and 4. Moreover, this was borne out by the clinical picture. (Autopsies were not permitted.) It is the writer's impression that the outcome in some of these cases would have been a different one, if the disease had been arrested in the labyrinth.

The meningitis and abscess formation in the cases terminating in recovery (Observations 6, 7, 8 and 9) were caused by direct contact with diseased bone, without involvement of the labyrinth.

Judging from current experience with these lesions, it appears that meningeal inflammation by contiguity offers more favorable chances for a successful outcome after operation, than does meningitis subsequent to invasion of the labyrinth. The middle cranial fossa is more tolerant of the purulency and responds more promptly to its surgical evacuation, than the posterior cranial fossa. Gatherings in the last named location are not permanently relieved by surgical interferences, without preliminary control of the purulent process in the labyrinth.

In conclusion, I urge that expectant treatment in the face of post-mastoidal intracranial symptoms be limited as much as possible, and that as soon as diagnosed, or even a well-founded suspicion of purulent involvement of the meninges is entertained, that a systematic exploration of the meninges and labyrinthine capsule be undertaken.

It is not advisable to delay the operation on the labyrinth until the appearance of the meningeal symptoms. These will obscure the symptoms pointing to the labyrinth, and thus serve to diminish the patient's chances for ultimate recovery. A number of cases which would go on to general meningitis and death will be saved by the early establishment of meningeal drainage.

Exploratory operations are indicated in even the most desperate cases, from the clinical point of view. Remarkable

results have been known to follow, and precisely among the patients otherwise doomed to death, one or the other may have the scales turned in his favor, contrary to all expectations.

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III.

OCULAR SYMPTOMS OF INTRACRANIAL COMPLICATIONS IN OTITIC DISEASE.

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The progress of otology during the past ten years has been so largely practical and surgical that even the operations for intracranial complications have lost much of their originally formidable, not to say ominous, aspect. The otologist of today is, theoretically and technically, prepared, in case of definite indications, to follow up the suppuration from its source in the middle ear or mastoid whithersoever it may lead him. It is now generally recognized that complicated methods of localization and the use of base lines, measurements and cranial topographic studies are of minor importance, as we have learned that the intracranial pus accumulations are, in a majority of cases, to be found in the immediate neighborhood of the purulent infectious focus in the temporal bone, or at least that they can most easily be reached, treated and drained by an extension of the original field of operation. While this increase of security in operating and the consequent improvement in surgical statistics render the question of diagnosis less immediately vital and important, they by no means lessen its scientific import or make it any less incumbent to place upon a definite basis, as far as possible, the question of the significance of intraocular changes in the diagnosis and prognosis of endocranial complications of purulent otitis.

An interesting feature in the study of ocular symptoms in otitic intracranial complications and one of importance in the history of investigations on this subject is the fact that in this country many ophthalmologists are aural surgeons as well. A generation ago, when the surgery of otitic disease was in its infancy, this was almost invariably the case, and it is due in large measure to the work of Kipp, Gruening, Knapp, Roosa, Alt and other Americans that the teachings of Politzer, Bezold, Zaufal, Lucae, Troeltsch and Schwartze have been spread in this country. Kipp was, in fact, the very first to

call attention to the importance of making ophthalmoscopic examinations in cases of ear disease. His first article¹ on this subject antedated that of Zaufal by a year, yet the latter is always spoken of, at least by German authors, as the pioneer in this field. Kipp's second article² appeared in 1885, and the conclusions as to the value of ophthalmoscopic changes in prognosis and treatment stated in his second paper are now generally accepted. Hansen urges the importance of the otologist being skilled in ophthalmoscopy in order to insure early and repeated examination of each and every case. American otology recognized and fulfilled this requirement years before it was formulated by Hansen in Germany.

Diagnostic signs are of practical import, whatever their scientific and theoretic value may be, as far as they serve as a basis for therapeutic considerations, prognosis or operation. This does not imply that a study of symptoms in cases of diseases which for other reasons are beyond the reach of therapeutics, at the time, is lacking in academic interest or in eventual practical importance, as the progress of methods of treatment may finally come abreast of the diagnostic methods. In the sphere of brain surgery or that part of it associated with the intracranial complications of otitic disease, the practical indications to explore the cranial cavity and determine the condition of sinuses, meninges and central organs are quite definite and generally urgent. The making of an accurate diagnosis hardly aids in treatment in this field. On the other hand, there is a development of other diagnostic methods, among which we may mention lumbar puncture, the blood examination and differential leucocyte count, the careful examination of the reflexes, Kernig's and Babinski's signs, the careful bacteriologic examination of aural discharge and of tissues removed from mastoid, sinus or jugular vein at the time of operation.

Inflammatory changes or circulatory disturbances in the optic nerve have always been considered corroborative evidence of similar endocranial conditions causing an increase of brain pressure or indicating inflammatory processes. In the otitic complications the evidence is often contradictory, obscure or insufficient. The views of the most experienced observers and the opinions expressed by standard authors on this point do not agree perfectly.

Bacon.³—Temporosphenoïdal abscess. Optical aphasia a

symptom of the highest diagnostic value. The patient, when shown an object, knows it, but is unable to name it (better, has no conception of its name; the other phrase might mean that he could not speak the name he had in mind, i. e., motor aphasia), but he is able to recognize and understand its name, and will often describe the object. He can sometimes name the object if allowed to handle or smell it (showing there is no motor aphasia, and that a conception of name, due to stimulation of memory pictures, can be accomplished over other paths than the visual). In one case there was, besides the difficulty in naming an object, difficulty in calling to mind its appearance, visual amnesia (Starr), "due to a suspension of function in association tracts lying deep in white matter of temporo-occipital lobes."

Inflammation of optic nerve may or may not be present. More frequent in cerebellar abscess. Symptoms of latter may be simulated by disease of semicircular canals. In the case of a child with suspected cerebellar abscess characteristic symptoms, including choked disc with retinitis, were entirely abolished by removal of granulations from the middle ears and establishment of proper drainage. In an abscess of the right cerebellar hemisphere there was edema of the right optic nerve. Photophobia and nystagmus may occur.

Sinus Thrombosis.—When the clot has extended to cavernous sinus, and ophthalmic vein has become engorged, there is likely to be a certain amount of edema of the eyelids on the corresponding side. Neuroretinitis is noticed in some cases, more especially in the later stages, but it is not constant.

Purulent lept meningitis. Photophobia, pupils contracted and responding but little to light, and optic neuritis. Later the pupils are dilated and often unequal. In children, symptoms suggestive of meningitis may be due to retention of pus in the middle ear. When paralyses occur, and there are optic neuritis and other characteristic symptoms, the diagnosis is assured. (Koerner does not lay much stress on neuritis in meningeal irritation in children, and cites a case in which he was urged to operate on account of double choked disc. It was a serous meningitis of mild type, and, as pulse and general condition were good and weight was increasing, he temporized. Recovery was spontaneous.)

Blau.⁴—It is advisable to consider any disturbance of ocular motility occurring in the course of an otitis media as sugges-

tive evidence, symptom of secondary involvement of the labyrinth or of intracranial complications. For the diagnostic value (*Verwerthung*) of ocular symptoms in the diagnosis of intracranial complications, it is of prime importance to determine whether morbid changes, particularly of the fundus, can be induced by middle ear disease. Cases cited to substantiate such a possibility were all suspicious of already existing complications, and were so considered by the observers themselves. Ocular symptoms are, then, of definite significance, to the extent that they appear to develop only when otitis has involved the interior of the skull, with the exception of nystagmus, which may be excited as a reflex on irritation of the "tonus labyrinth" (semicircular canals). Hyperemia of the nerve, neuritis, choked disc, render it highly probable that we have to deal with threatening or existing intracranial complication, but do not justify any conclusions as to their nature. On the other hand, the absence of intraocular changes by no means excludes the presence of intracranial suppuration, so that in the presence of other characteristic symptoms the negative result of ophthalmoscope has absolutely no significance. It is of importance as corroborating the other symptoms and clinching the diagnosis when present.

Burnett⁵ quotes T. R. Pooley to the effect that "if the optic neuritis is found in connection with purulent inflammation of the ear, the diagnosis of extension of ear disease to the brain is certain, no matter whether other evidence exists or not." Unfortunately it does not explain the nature of the intracranial lesion, and, while optic neuritis in chronic otorrhea, or even acute otitis, would be an urgent indication for immediate mastoid opening, its occurrence must be considered only in connection with other symptoms as an indication for an exploratory opening into the cranium.

Optic neuritis is usually not observed, or only slightly marked, in uncomplicated sinus thrombosis; it is sometimes found with extradural abscess. In cavernous sinus thrombosis, due to the passage of infection from the middle ear by the carotid canal (Koerner) edema about brow and orbit, exophthalmos, paralysis of extraocular muscles, immobility of the globe (due to orbital cellulitis), edema of lids, chemosis of conjunctiva and choked disc on the side of the chronic aural suppuration, are seen.

In otitic cerebral abscess and in extradural abscess neuritis

is frequent. Optic acoustic aphasia is usual in abscess of left temporal lobe, or in the right lobe if the patient be left handed. Ptosis of left upper lid has been seen in left temporal lobe abscess (Steinbruegge). Sensory aphasia with homonymous hemianopsia with preservation of pupillary reaction points to temporal lobe.

Symptoms of extradural abscess or of sinus thrombosis always dominate and mask those of brain abscess. This applies to fundus changes also.

Cerebellar abscess shows varying pupils, the wider being on the side of the abscess, conjugate deviation away from affected side, horizontal nystagmus, abducens paralysis, lagophthalmus, due to facial paralysis. Double optic neuritis is often the only symptom, but the fundus may remain normal even in fatal cases⁶. Cerebellar abscess is very likely to obstruct circulation of cavernous sinus and ophthalmic vein.

Meningitis.—No mention of ocular symptoms.

Bezold.⁷—Labyrinthine suppuration shows, at least in early stages, characteristic symptoms of irritation, subjective dizziness, vertigo even while lying down, disturbance of gait, horizontal nystagmus when gaze is directed to the sound side, and vomiting. Nystagmus may be rotary and always disappears (destruction of ampullar organs) sooner than the dizziness. Often complicated by fatal meningitis and cerebellar abscess.

Extradural pus collections may give no clinical signs.

Operate after eight weeks in acute otitis, and after several months of fruitless treatment of fetid discharge in chronic cases to find the source of continued suppuration.

Choked disc and double edema of both lids speak for thrombosis of both cavernous sinuses.

In brain abscess, optic neuritis, oculomotor or abducens paralysis. Otitic leptomeningitis may start from any point of a sinus thrombosis, or from brain abscess which has ruptured into ventricle or subarachnoid space. Extradural abscess may cause necrosis of dura and spread to arachnoid and pia.

Dench. Serous meningitis (1903).—Most frequent paralyses those of the third, fourth and sixth nerves. Paralysis of the third nerve causes at first contraction and later dilatation of the pupil. An early symptom is failure to respond to light. Where local paresis exists, divergent strabismus is probably the most frequent symptom. The fundus will most generally show either a choked disc or an incipient swelling of the optic

papilla. It is strong corroboration of the other symptoms of meningitis, but is found in any intracranial lesion which increases pressure. Sinus thrombosis. Presence of choked disc will confirm the diagnosis in doubtful cases.

Brain abscess.—Choked disc may be present, but is not characteristic of abscess alone.

Cerebellar abscess.—Choked disc is often present.

Brieger.⁸—Fundus changes are generally missing in uncomplicated extradural abscess, and arouse suspicion of other endocranial complications. Extradural compression causes disturbances in fundus which are rapidly compensated. Repeated examination may detect transitory fundus changes. Abducens paralysis is not infrequent. Kessel reports paralysis of abducens and oculomotor nerve of the opposite side, due to collateral edema of meninges or brain, and not to direct compression.

Purulent leptomeningitis. While Pitt found the fundus normal in all uncomplicated cases and while Knies claims optic neuritis as the most valuable diagnostic aid, the truth lies in the middle. His experience agrees with that of Hansen, that about half the cases show changes of the fundus which are sufficiently marked and unambiguous to allow a diagnosis. But these changes never appear as typical choked disc. They are rather in the nature of optic neuritis, and vary from slight hyperemia to entire obscuration of the margin of the disc. Generally double, occasionally unilateral, or unequal in degree, or even more marked on the side of the sound ear. In the cases with insidious onset optic nerve changes were rarely seen at an early stage before manifest symptoms developed. The pupils show a markedly rapid variation between contraction and maximum dilatation.

Abducens and oculomotor paralysis are not always due to localization of the purulent process at the base, but may be due in still circumscribed meningitis to general increase of pressure in the cerebrospinal fluid and be present as the only symptom for some time before the meningitis becomes generalized.

Nystagmus may be present in the form characteristic of labyrinthine disease or, more correctly, of implication of the semicircular canals, i. e., horizontal oscillations when the gaze is directed toward the sound side. After generalization of the purulent process, and not infrequently after operation, the nystagmus also becomes generalized in the sense of occurring

with all ocular rotations, either constantly, and even when the eyes are at rest or the lids closed, or only when an object is fixed. This would indicate a spreading of the suppuration from the horizontal to the other two semicircular canals, independent of movements of the eye.

Conjugate deviation is rare.

Serous meningitis. Eye symptoms, as neuritis of slight degree, never as choked disc, with only slight hyperemia of the papilla. May be transitory or develop early, but may also appear later (Hegner) even several days after increase of intracranial pressure had been relieved by operation. Abducens paralysis may be the only symptom of serous meningitis locally, due to compression at the base. A case of nuclear lesion on the floor of the fourth ventricle, causing paralysis of associated motion (*Blick-laehmung*) has been reported by Brieger.

Kirchner.⁹—Cavernous sinus thrombosis may develop early not only in the chronic but in acute otitis media, especially after influenza. Exophthalmus, edema of the lids, paralysis of extraocular muscles, disturbance of vision, are the usual symptoms.

Kyle, J. J.¹⁰—Purulent leptomeningitis. Contraction of pupils, loss of light reflex, later dilatation.

Thrombosis. Optic neuritis is one of the symptoms (quoted from Ballance). Extradural abscess. Occasionally optic neuritis a suggestive symptom.

Love, J. K.¹¹—Meningitis. Pupils contracted, reaction to light lessened. Later, optic neuritis is often present, pupils unequal and dilated. Ptosis and squinting may occur.

Sinus thrombosis may simulate meningitis. Optic neuritis may be present. Brain abscess. In early, irritation stage, pupils contracted, photophobia. Later unequal and do not respond to light. In temporosphenoidal abscess, third nerve may be involved, and ptosis, accommodation, paralysis and oculomotor paralysis of all but external rectus (abducens) occur. Optic neuritis occurs in about half the cases of cerebral and 60 per cent. of cerebellar abscess.

Koerner.¹²—Extradural abscess. Neuritis not unusual (Pitt, Lane, Johnson, Zaufal). Of 11 uncomplicated cases, 8 normal. Of 20 uncomplicated in Schwartze's clinic (Grunert), all normal. Koerner cites two cases, however, of marked changes, both in children, with extradural abscess in posterior fossa. In one case the neuritis increased after evacuation of

the abscess, particularly on the sound side. In the other, double choked disc, also more marked on the sound side, first appeared after operation. Cure in both. There was a possible complication with sinus affection in both.

Knies' view as to the importance of optic neuritis in diagnosis of meningitis is not shared by Koerner. The cases of Kipp and of Andrews were not subjected to autopsy, and the histories read like complication with sinus phlebitis and extradural abscess, respectively. In serous meningitis, particularly in children, optic neuritis has been observed in light as well as in severe cases. This is the more surprising, as neuritis is missed so often in purulent meningitis.

Thrombosis, neuritis and choked disc common in cavernous sinus thrombosis. Koerner has not seen it in sigmoid sinus thrombosis, uncomplicated. Leutert saw neuritis in 1 of 10 cases, Hansen, 3 in 8 uncomplicated cases. Jansen finds the changes often and finds them of diagnostic value.

Jansen has seen nystagmus in sinus thrombosis and extradural abscess.

Brain abscess. Neuritis frequent, but not invariable. Generally double and more marked on the side of the abscess. Late symptom and not marked, and may advance after evacuation of the abscess. Sight often but slightly affected, and consecutive atrophy rare. Hansen, 12 cerebral abscess, 2 with vessel changes and 4 with optic neuritis; 7 cerebellar, 2 optic neuritis. In one of the latter, neuritis only on side of abscess, in 3 double and equal, in 4 more marked on abscess side.

Total amaurosis seen in 3 cases by MacEwen and Berridge, reported in detail by Koch (*Otit. kleinhirn Abscess., Berl., 1897*). Operation in one case, with disappearance of amaurosis. In one fatal case there was complicating ventricular serous meningitis and purulent leptomeningitis. Case 3, autopsy incomplete. Nothing similar has been observed in cerebral abscess.

Hemianopsia generally due to lesion of the internal capsule, or when situated in occipital lobe to subcortical lesion of the optic radiation. These are distant symptoms due to pressure of abscess on neighboring regions and edema of brain substance.

Oculomotor palsy frequent. Temporosphenoïdal abscess of at all considerable size invariably causes paralysis of some side, generally incomplete and limited to pupillary fibers and levator

(ptosis and accommodation paralysis), occasionally involving all branches.

Pupillary mydriasis common in cerebellar abscess.

Abducens paralysis may be homolateral or crossed. It is of little diagnostic value, occurring in serous meningitis and spontaneously healing complications.

Conjugate deviation may be toward side of abscess. (Kalmus, Moos) or away from it (Schwartz, Acland and Ballance, Hansberg, Winter and Deansley). Nystagmus (Mueller, Winter and Deansley, Ballance, Barling, Okada). It is a question whether this is not a labyrinthine symptom.

Partial or complete oculomotor paralysis is a valuable sign of temporosphenoidal abscess.

In children, brain tumor (tubercle) not infrequently complicates suppuration in the temporal bone. Choked disc speaks more for tumor, neuritis for abscess. Chorioidal tubercle may be discovered.

Knapp, H.¹³—Pachymeningitis externa; epidural or extradural abscess. Symptoms of meningitis are present in irritation in many cases—headache, slight rise of temperature, choked optic disc, pressure pain, somnolence, slowing of pulse, stiffness of neck, but these symptoms are too indefinite to make a diagnosis.

Pachymeningitis interna, subdural or intradural abscess with softening and abscess of adjacent brain substance—circumscribed encephalitis—occurs when pia and arachnoid are agglutinated over inner surface of an infected area of the dura and exudation or perforation takes place. If not adherent purulent leptomeningitis results. Optic neuritis is rare.

Sinus thrombosis and pyemia. As the last of eight symptoms in importance and frequency, Koerner mentions stupor and optic neuritis as present only in complication with encephalic abscess and meningitis.

Brain abscess, four stages, initial, manifest, latent, terminal. Optic neuritis, earlier and more pronounced on the diseased side, is one of the symptoms of brain pressure.

Homonymous hemianopsia, on record seven times, probably would have been found oftener if looked for regularly.

Partial or total paralysis of the motor communis oculi and abducens nerves, conjugated deviation of the eyes (to either side), and nystagmus, are rare and indefinite symptoms.

Lake, R.¹⁴—Meningitis. In progressive cases, intolerance of

light, at times amounting to photophobia, becomes marked. The pupils, which are commonly unequal, react to light, but usually more or less slowly than normal. Optic neuritis or papillitis will in most cases be noted. After development of the lethargic state the pupils often become widely though unequally dilated, and all reaction to light is gradually lost, while in basic cases a tendency to squinting is more frequently noted. Nystagmus may be present in later stages.

Extradural abscess. It is rare to find optic papillitis in uncomplicated cases.

Brain abscess. Pupil on affected side frequently contracted and only reacts sluggishly to light and accommodation, in which case the abscess will be found to be small, relatively, but if it is of large size the pupil on the same side will be both dilated and stable. Paralysis of the third nerve on the same side as the abscess is a variable sign; optic neuritis, or papillitis, is found during the whole of this stage and may be only on the affected side.

Cerebellar abscess. In second stage there is not infrequently conjugate deviation of the eyes to the side opposite to the lesion, along with optic neuritis. Lateral nystagmus with large excursions is especially frequent in lateral lobe abscess, the movements of the eyeball are toward the side on which the abscess is situated. Rotary nystagmus points toward involvement of the middle lobe. (These considerations seem to be largely hypothetical. If the movements of the eyeball are toward the side, any side, there must be deviation, if not, there is merely horizontal nystagmus. Lake may mean that nystagmus develops only when the eye looks toward the affected side.—P. H. F.)

A final decision on such points is often impossible until a large number of cases has been collected, critically studied, classified and analyzed and a statistical study made of the results. Statistical studies of this sort have been made in Germany by Sessos, Hansen, and by Koerner, whose cases were supplemented a year or two later by Takabatake. In this country there have been no analyses of large series of cases, although various otologists have reported their observations of specially interesting, grave or successfully treated cases of brain abscess, meningitis and sinus thrombosis.

Hansen's statistics comprise 97 cases, of which 45, about 46.4 per cent., showed optic nerve changes, while Koerner reports 54 cases with positive ophthalmoscopic findings in 16, or 29.6

per cent. Sessous (Lucae Festschrift, 1905) discusses 104 cases, 5 per cent, of all those treated at the Berlin clinic from 1895 to 1905. Ophthalmoscopic changes were most frequently noted in meningitis complicated with sinus thrombosis, least often in extradural abscess. A normal fundus is no proof of absence of intracranial complication, nor does the presence of fundus changes allow us to draw any but the most limited and cautious conclusions as to their presence. This also holds true in regard to prognosis as to recovery. Fundus changes are of diagnostic importance only when other cerebral symptoms are lacking. This is in marked contrast to the view of Ostmann cited above, that fundus changes clinch a diagnosis already made probable by other symptoms, but are of no value by themselves, and to the opinion of Koerner and of Hansen, who attach great importance to positive findings in the ocular fundus in suspected cases of intracranial suppuration. Hansen sums up his observations as follows: "Fundus changes may occur in any intracranial complication. Least frequently (18 per cent.) in extradural abscess, and then more often in the deep seated than in the perisinuous variety, they are most often observed, 87.5 per cent., in cases which had been considered as hyperemia of the brain and meninges, or serous meningitis, in 57 per cent. of uncomplicated, and 47 per cent. of complicated purulent meningitis (about 50 per cent. of all meningitis), in 50 per cent. of cerebral, and 29 per cent. of cerebellar abscess, 37.5 per cent. of uncomplicated and 47.5 per cent. of complicated sinus thrombosis (agreeing with Jansen). In other words, fundus changes are about equally frequent in the various forms of otogenous intracranial suppuration, occurring in about 44 per cent. Neuritis is the most common condition, slight neuritic change, vessel changes on the disc next in frequency, while real choked disc is unusually rare (3 per cent.) and occurs only under certain definite conditions. Both eyes were affected in the great majority (80 per cent.) of positive cases, and the changes were usually (75 per cent.) more marked on the affected side, although the opposite condition was noted in 9 per cent. Unilateral involvement of the fundus, observed in 20 per cent. of the positive cases, was invariably situated on the side of the ear affection. In brain abscess the fundus changes were almost invariably (87.5 per cent.) bilateral, and more marked on the affected side (50 per cent.) or of equal intensity (37.5 per cent.). Hansen does not agree with Koerner, MacEwen and

Oppenheim that fundus changes in brain abscess are not infrequently found on the side opposite the intracranial lesion. As other symptoms of intracranial complications are often variable, evanescent, scanty and obscure, and the recognition of these ominous conditions consequently difficult and uncertain, positive evidence of fundus changes, which can always be detected when present (except in cases of opacity of the media, as cataract, etc.) is of undeniable and decided value. Their development in otitis suppuration always indicates the presence of some intracranial complication, for it is an unproven and improbable assumption that middle ear disease alone, without endocranial suppuration, can induce morbid changes in the optic disc. As such changes are found in only about one-half of all cases of intracranial complication, their absence is never to be considered as positive evidence. Fundus changes are of definite importance in suppurative otitis, as their development, even in the absence of all other cerebral symptoms, indicates some intracranial complication, and accordingly a more severe affection. The prognosis of intracranial complication itself, on the other hand, is not influenced by the presence of fundus changes, as the latter may occur in the least dangerous as well in the most serious forms, and in mild as well as in severe or even fatal cases of a single form. Ophthalmoscopic examination should be made in every case of otitis suppuration, and repeated frequently in case there is any unusual feature in the progress of the case or even the slightest cerebral disturbance. The otologist should carry out these examinations himself, as this is the only guarantee for their regular methodical performance and record. Disc changes are an immediate indication for opening the mastoid in a case of middle ear suppuration which for any reasons has not been operated. The subsequent course of the case must then decide whether further surgical intervention is necessary.

Koerner agrees in the main with Hansen as to fundus changes being bilateral, even when the intracranial disease is limited to one side, in the great majority of cases, and as to the presence or absence of fundus changes having no significance for the prognosis of the intracranial complication. In purulent meningitis he comes to quite different conclusions, all of his 6 uncomplicated cases having had normal optic disc up to the time of death, while of the 8 complicated cases 6 showed fundus changes. Simple thrombosis caused disc changes in

only 1 of 5 cases. One-sided occlusion of the cranial sinuses, it seems, is not sufficient to produce venous congestion in the eyeground, and this applies as well to the sudden disturbance of circulation incident to jugular ligation as to the more gradual stoppage by thrombosis or endophlebitis. Only about 25 per cent. of all cases of intracranial suppuration showed optic nerve changes. They are more generally absent than present, and more apt to indicate a combination of conditions than any one complication. Thus of 18 cases of a single complication only 3 showed fundus changes, and even in these there was some suspicion of another form of intracranial suppuration. Koerner sums up as follows: The variety of fundus change, neuritis and choked disc, respectively, does not allow any conclusions as to the character of the intracranial complication. A more marked development of fundus changes on one side does not indicate exclusive or even predominant involvement of the corresponding half of the cranium. Fundus changes do not influence the prognosis, and even advance of these changes after evacuation of pus in the cranial cavity does not alone make the outlook any worse.

My statistics and conclusions are based on a study of the records of the New York Eye and Ear Infirmary from 1905 to 1907. Of a total of 19,129 cases of ear disease treated during these two years, 11,176 were affections of the middle ear and Eustachian tube, 1,064 affections of the mastoid. There were 11 cases of cerebral, 25 of epidural, and 73 of subperiosteal abscess; 12 of jugular vein and 13 of lateral sinus thrombosis. These figures represent the cases so diagnosticated on admission. The full number of complicating intracranial cases is to be determined only from the report of operations which is shown on Table 1, which shows 16 exploratory and 65 curative operations for sinus thrombosis, 24 exploratory and 6 curative for brain abscess; 31 ligations of the internal, and 1 of the external jugular, and 2 extirpations of the internal ear for labyrinthine suppuration. These cases were operated on by or in the service of Drs. Dench, McKernon, Bacon, Whiting, Lewis, Adams, W. S. Bryant, Rae, J. D. Richards, Stevens, Kenefick and Brandegee.¹⁸

A study of these records shows that the great majority of cases of intracranial otitic complications were examined at some time or another during the stay in the institution, for evidences of changes in the fundus oculi. A few cases were

not so examined, either on account of death having come on suddenly a short time after admission, or because the classic symptoms of intracranial disease assured the diagnosis, or finally because the symptoms receded immediately after operation. These reasons are of practical weight, but from a scientific point of view it is to be regretted that systematic examinations of the fundus are not made in every case of operative ear disease. The presence of choked disc or of an optic neuritis several days after operation is open to several interpretations in regard to the period of development of the causal complication, whereas its significance is very much less ambiguous in case a normal fundus was found at a previous examination. Koerner lays stress, and, justly, on the importance of repeated examinations "up to the time of cure or death, i. e., until the diagnosis has been established, etc." The corroboration of the ophthalmoscopic findings, or rather of the diagnosis which they are to assist, is unfortunately beset with more than usual difficulty. In the case of recovery the character of the ailment is often no more positively determined than it was before, or, at all events, there is no certainty of such degree as to enable us to stamp the diagnosis as agreeing with the intraocular changes or not. The findings at operation may be of value, especially in the event of positive evidence, in the form of the evacuation of pus, of a brain abscess, of an occluded, thickened or ulcerated sinus, discolored meninges or subdural abscess formation, but in other cases they fail to clinch the diagnosis. The only absolute verification, namely, by examination post-mortem, is excluded, of course, in case of recovery, and frequently by the inability to obtain permission for autopsy. This is the rule rather than the exception, and unfortunately it is just in the most serious cases, those dying of cerebral complications, that an addition to our definite knowledge would be of greatest value. From this point of view our statistics, no matter how large, are much less instructive than the smaller ones of continental, and particularly of German, observers, who have been enabled in the great majority of cases to corroborate or disprove the *intra vitam* clinical diagnosis by autopsy and pathologic examination post-mortem. The postulate of examination by experienced and skilful ophthalmologists is fully met in the case of the institution cited in my study, as it has a large and active eye department, and numbers among its surgeons Gruening, Bull, Derby,¹⁸ Callan, Weeks, Marple and other well-

known oculists. The internes, too, have with few exceptions had a double service, with ample practice in the use of the ophthalmoscope and the study of fundus conditions both in the in- and out-patient department. In many instances the name of the examiner is mentioned, and in some, several consultants have expressed their opinions as to the fundus conditions. Where no examiner is specified the ophthalmoscopic findings were made by the house surgeon. In case of doubt or of unusual conditions the attending surgeons were called on for an opinion. As in many other reports of fundus conditions accompanying otitic complications, it appears that there is not sufficient attention paid to the necessity of a strict differentiation between optic neuritis and choked disc, or, in the case of already established atrophy, to the distinction between the simple and the postneuritic form. While in the latter case it may not always be easy even for the experienced ophthalmologist to detect slight traces of previous inflammation about an atrophic disc, such as irregularity of outline, proliferation or irregular distribution of pigment along the disc margin, filling up of the lamina cribrosa with connective tissue, irregularity of lumen, thickening of vessel walls or connective tissue striæ in their course, some or most of which changes may be wanting, there is usually no doubt at all, in a specific concrete case, whether we have to deal with neuritis or choked disc, at least in the early stage. And here, again, is a reason for early as well as for repeated ophthalmoscopic examinations; a choked disc may be engrafted or superadded to a neuritis, or *vice versa*, in a case, respectively, of an intracranial inflammation, such as meningitis being complicated by a process which greatly increases brain pressure, as, for example, brain abscess or internal hydrocephalus, or, on the other hand, in the still more unusual event of inflammatory changes in the meninges occurring in brain abscess or thrombosis.

The scope of this presentation does not permit of a detailed consideration of the pathogenesis and mechanical development of choked disc and optic neuritis, a subject of practical import for the interpretation of clinical pictures in cerebral disease, particularly as there is still a difference of opinion as to the exact manner in which these intraocular conditions are brought about, and a number of conflicting theories to explain them. We may in a general way accept the prevalent opinion that choked disc is due to embarrassment of venous off-flow from

the globe due to pressure on the nerve either by sheaths distended with inflammatory fluid exudate, solid mass, or edematous transudate, or to edema of the nerve itself, as from neuritis, and is accordingly purely or mainly of mechanical origin, while neuritis may be due to circulatory disturbance, to bacterial infection, to the action of toxins working at a distance from a purulent focus, to mechanical compression as in choked disc, or to a combination of some or all of these factors. For the practical purpose of the otologist choked disc signifies a morbid process which has resulted in a compression of brain tissue or at least in diminution of free space and so to a rise in intracranial pressure, while optic neuritis points to an inflammation of or in the immediate neighborhood of the meninges and an extension of this process either by continuity, or more probably by circulating bacteria or toxins, to the sheaths, and eventually to the substance, of the optic nerve.

The two conditions are dissimilar in symptomatology, clinical course, and to a certain extent in prognosis. Choked disc is characterized by protrusion, often amounting to several diopters, marked edema, generally marked dilatation or tortuosity of the veins, with narrowed arteries. Retinal hemorrhages are not infrequent. The nerve fibers become swollen and the nerve head appears made up of radiating striæ, often flecked with linear hemorrhages and resembling a chrysanthemum. There is no marked irregularity or obscuration of the disc margin. In extreme cases, of course, the intense congestion may induce slight inflammatory changes so that there may be a mild neuritis in addition. After relief of excess pressure in the cerebrospinal fluid a marked degree of choked disc may entirely disappear. Atrophy is an unusual sequela and is usually attributable to complicating neuritis or to intercurrent disease of the nerve or descending degeneration or inflammation from central disease. In neuritis, there is usually little or no swelling of the disc, so that protrusion is not noted. Hyperemia, on the other hand, is marked, and the outlines of the disc soon become indistinct or entirely invisible. This in marked contrast to papillary stasis, in which the disc is often enormously swollen and prominent, yet distinctly visible. The obscuration of the disc in neuritis may be so marked that the position of the nerve head is merely indicated by or rather to be inferred from the converging vessels at the center of the fundus. Marked changes in the caliber or course of the vessels are infrequent.

almost exceptional. On the other hand, we note changes in the vessel walls in the form of perivascular striations, connective tissue proliferation starting at the disc and accompanying veins and arteries out along the fundus, as well as deposits of fibrin in the physiologic excavation of the disc, i. e., in the lamina cribrosa as well as at the margin. Of course, these changes are often obscured by the intense hyperemia and inflammation of the disc and by a fine opacity of the posterior layers of the vitreous and edema of the central part of the retina which almost invariably accompanies this condition. It is only in the later stages, particularly that of postneuritic atrophy, a frequent sequela, that these processes are made manifest by irregular outline of the disc, blocking up the lamina and physiologic excavation, and veiling of the markings of the cribrosa, perivascular connective tissue strands, and by pigment deposits on the disc or along its margins. Retinal hemorrhages are rare in uncomplicated optic neuritis. When present they may be due to choked disc, to vessel wall changes caused by mechanical obstruction of the circulation, or to vascular disease of a septic nature, as in pyemia or metastatic or septic retinitis.

In the construction of the table I have spoken, quite generally, of fundus changes, as opposed to the normal eyeground, in order to render the comparison of positive and negative findings more simple and graphic. In each case, however, the fundus conditions, whether neuritis or choked disc, and their distribution in one or other fundus or both, whether equally, or predominantly on one side, are indicated by the signs which are explained in the footnote. In the same table the cases are arranged in two main divisions, according as they resulted in death or recovery, so that it is possible to compare the frequency and severity of the fundus changes in mild and in fatal cases for every variety of intracranial complication. This, it seems to me, makes it much easier to get a general view of the subject than if we follow the example of Hansen and of Koerner and construct a number of tables dealing separately with the factors of distribution, severity, frequency and result. We are better able to judge of the prognostic significance of the presence or absence of fundus changes in endocranial complications if each form of disease is shown separately than if we make two categories, death and recovery, and compare the fundus findings in the two divisions for the entire number of cases. Instructive facts may be obscured by such figures which are actu-

ally out of place. For it is evident that figures which show a remarkably high percentage of fundus changes in fatal cases of, let us say, sinus thrombosis, will be balanced and lose their identity if combined with others showing indeterminate, negative or even opposite conditions in abscess or meningitis. The same considerations apply to a statistical study of the distribution of fundus changes, meaning the occurrence in one or other eye with greater frequency or severity, and the significance, if any, of bilateral changes.

A study of the individual and various forms of intracranial complications does not bear out, in my statistics, the conclusion of Koerner that the presence or absence of fundus changes is of no value in the prognosis of intracranial complications. The results as to distribution are also less ambiguous. Koerner found intraocular changes, without exception, in every instance, bilateral, and more often to a more marked degree on the sound than on the affected side.

EXTRADURAL ABSCESS.

In 6 cases of uncomplicated extradural abscess the fundus was invariably found normal. In 8 cases complicated with sinus thrombosis there were fundus changes in 3 (37.5 per cent.), all bilateral.

MENINGITIS.

In purulent leptomeningitis without other complications, changes of the nerve head were found in 4 out of 5 cases, all of which ended fatally. The changes were bilateral in all but one case, in which they were limited to the side corresponding to the mastoid involvement. In this case, choked disc was found, which in another was associated with optic neuritis of the other eye. Of 9 complicated cases neuritis was found in 6, slight changes in the vessels in 1, and a normal fundus in but 2. The intraocular disturbance was without exception bilateral.

SINUS THROMBOSIS.

In sinus thrombosis fundus changes were found much more frequently than might have been expected from the reports of previous statistics. Of 17 uncomplicated cases, no less than 12 showed decided fundus changes. All the fatal cases, 12 in number, showed fundus changes, while the 5 without fundus

changes all recovered. In these cases, too, the intraocular disturbance was always bilateral, with the exception of the case reported by Gruening and included in my statistics, already mentioned, and one other, in which a thrombosis of the central retinal vein, probably of metastatic origin, was the only fundus change. In sinus thrombosis with extradural or perisinuous abscess, of 7 cases which recovered only 2 showed neuritis. Of the 2 fatal cases, one was normal, the other showing double neuritis. In all of this group, again, the fundus changes were bilateral, except in a case of general thrombosis of the sinuses complicated with meningitis on one side only, strange to say, and it was a question to which of the two intracranial complications the intraocular condition was to be attributed. The other cases of complication with meningitis are grouped under the latter heading above. Of the entire series of thrombosis cases, 28 in number, changes were found in 17 (61 per cent.). This observation is in marked contradiction with the figures of Hansen, who found them in only 37 per cent. of his thrombosis cases, and of Koerner, who had 5 uncomplicated cases, all but one with normal fundus. In view of these figures we must modify Koerner's conclusion that unilateral occlusion of the venous channels in lateral sinus and jugular does not seem sufficient in most cases to cause congestion in the ocular fundus.

BRAIN ABSCESS.

One case of uncomplicated temporosphenoidal abscess and 3 of uncomplicated cerebral abscess showed normal conditions in the fundus. In one case of uncomplicated brain abscess there was neuritis. Reviewing the combination of observations thus briefly reported, we may still say that changes in the fundus oculi may be absent in all and any forms of endocranial affection, but, and this is almost as unfortunate from the standpoint of differential diagnostics, they may also be present in any and all, with the possible exception of uncomplicated extradural abscess. An exclusive or more markedly unilateral ocular involvement is rare and generally without significance, but in sinus thrombosis it may be a valuable sign. The prognostic significance of fundus changes for the course of the intracranial complication is somewhat obscured by the fact that mild cases are often not examined ophthalmoscopically, while in the fatal cases the condition of the fundus is more apt to have been investigated at some time or another. It would appear that the

absence of neuritis in thrombosis is of good omen, as it was not missed in a single one of my fatal cases, but the presence of neuritis does not make the outlook more grave, as there is about an equal percentage of recovery and of death after it has been found.

Dr. J. D. Richards, of New York, has been kind enough to give me the results of his own experience in this field. He has seen extradural abscess produce papillitis in three cases, in all of which it was on the affected side. What percentage this is he is unable to state, but thinks it must be exceedingly small. In sinus thrombosis it is probably not over 5 per cent. Richards has not seen the high percentage referred to by some writers as associated with thrombosis. A point of interest in the sinus cases to which Jansen and he have called attention is that those cases which showed neuroretinitis had the jugular thrombosed. In these cases the inferior petrosal is also blocked and the damming back of venous blood upon the cavernous sinus and thus upon the ophthalmic vein and intraocular vessels is probably responsible. He believes that when we have a sufficient number of observations to enable us to draw definite conclusions, this agency of jugular bulb thrombosis in the causation of fundus changes will be corroborated. At least three of the cases collected by me seem to support this view. In one (43) there was a thrombus in the lower part of the sinus only, probably implicating the bulb, and double optic neuritis. In another (67), ending fatally, there was neuritis on the thrombosed side and later positive evidence of cavernous sinus involvement in the form of edema of the eyelids, beginning on the affected side and afterwards involving both eyes. In a third case (56), the sinus was apparently normal at operation. A peaked temperature curve, pressure pain along the jugular, and slight neuritis, more marked on the affected side, led to evacuation of the sinus and excision of the jugular vein, followed by recovery. In this case it would seem that there was a primary thrombosis of the vein, which at the time the mastoid was opened had not yet involved the upper part of the sinus. The conditions were almost paralleled in Gruening's case, which I have included in my statistics, as the fundus changes were found only on the affected side, and the sinus appeared healthy not only at the primary operation, but at the time of jugular extirpation. On incision, however, it was found to contain an infected clot, so that here, too, it seems quite probable that there

was a primary bulb thrombosis with very little tendency to advance upwards, but with rapid infectious softening.

Comparing the results of his own investigation with those of Hansen, Sessous and Koerner, the writer has but little to add, except in corroboration. The absence of fundus changes in a given case has no significance. Its presence, on the other hand, is of the greatest importance. This bearing is not strictly diagnostic, as we can not draw any conclusion from the presence of fundus changes as to the nature of the intracranial affection, but it is an absolute indication of the presence of some complication, and as such makes it advisable, if not imperative, to explore the cranial cavity and, if possible, remove the source of suppuration and of local as well as systemic infection. To put the case in a nut-shell, fundus changes may be absent in intracranial complications, but intracranial complications are never absent with fundus changes. The otologist should make his own ophthalmoscopic examinations. This does not imply that an ophthalmologist is not to be consulted in a doubtful case and the observations compared. Early and repeated examinations in each and every case can only be assured by the ability of the aural surgeon to use the ophthalmoscope with skill and experience. Ophthalmoscopic examination is usually neglected unless the previous history or the development of other symptoms of intracranial involvement directly suggest it. Early and repeated examinations would add greatly to the value of our statistics and to our knowledge of the pathogenesis and significance of fundus changes, and routine examination on admission of all operative cases would not only supply much valuable material, but avoid the loss of important observations due to the frequent impracticability of carrying out an ophthalmoscopic examination on the sudden development of ominous symptoms, the preparation for further urgent operation, or, indeed, sudden death.

In addition to the observations on fundus changes, I wish to report quite briefly on one or two other ocular symptoms. In a comparatively large number of cases of generally uncomplicated otitis or mastoiditis, edema of the eyelids limited to the affected side was found as an expression indicative of a marked involvement of the zygoma cells (1, 9, 15, 29), complicated in 2 cases (9, 29) by subperiosteal abscess. The same symptom was found in 3 cases of secondary inflammatory infection of the soft parts, after removal of all diseased bone (25, 26, 29)

in ordinary furunculosis of the external auditory canal (14, 63) and in stitch-hole abscess developing a week or two after operation (10, 23). In two cases, finally, this edema of the lids was due to conditions which had no connection or only an indirect connection with the otitis, as erysipelas (71) and frontal sinusitis (11). Lid edema as a symptom of intracranial complication was observed in 3 cases. Once in spontaneous recovery from a probably incomplete sinus thrombosis (21), again bilaterally in sigmoid sinus thrombosis with thrombosis or stasis in the cavernous sinus (67). Ophthalmoplegia occurred in 2 cases of brain abscess (2, 19), and, again, as an expression of mechanical fixation of the globe by inflammatory products in the orbit, in thrombosis of the cavernous sinus (38). Convergent strabismus was noted in meningitis (72), meningitis with sinus thrombosis (64), and meningitis with epidural abscess (32, 57, 59). Abducens paralysis occurred in one case of uncomplicated (?) middle ear suppuration, and once, probably as a result of traumatism, after exploratory incision of the brain (13), once in combination with paralysis of the trochlearis in a case of brain abscess with meningitis (30), and in one case, finally, of meningism following sinus thrombosis (55). Conjugate deviation of the eyes, as well as nystagmus, was found in several cases of brain abscess and meningitis, and in one of lateral sinus thrombosis, but, as the case histories and autopsy records, respectively, of these cases did not completely explain the symptoms or establish the diagnosis on a pathologic-anatomic basis, they will not be considered further.

BIBLIOGRAPHY.

1. Archives of Otolaryngology, Vol. viii, 1879, p. 147.
2. Ibid., Vol. xiv, 1885, p. 1.
3. Diseases of Ear, 1902.
4. Encyclopedie d. Ohrenh. Art., Auge, Ostmann, 1900.
5. Diseases of Ear, 1901.
6. Woodward, Arch. f. Otol., January, 1906.
7. Lehrb. d. Ohrenh., 1906.
8. Otogene Erkrank. d. Hirn., 1903.
9. Ohrenh., 1904.
10. Diseases of the Ear, etc., 1906.
11. Diseases of the Ear, 1905.
12. Otit. Erkrank. d. Hirns, etc., 1902.
13. De Schweinitz and Randall. System; Complications of Tympanic Inflammation, 1899.
14. Diseases of the Ear, 1903.
15. In addition to this material I have included all cases reported in the last (1906) volume of Transactions of the American Otological Society, of Intracranial Otitic Com-

plications in which an ophthalmoscopic examination was made. They are one case of lateral sinus thrombosis (Gruening), and two cases of cerebellar abscess (Dench, A. Knapp). Gruening's case was particularly interesting as there was bilateral mastoiditis with exposure of both sinuses at operation. Characteristic temperature changes arousing suspicion of thrombosis, an examination of the fundus was made and hyperemia of the discs found on both sides. On only one side, however, did this progress to optic neuritis, and on the strength of this finding, and in spite of the absence of evident local changes in the sinus wall of this side, and the presence of some discoloration on the other, the apparently healthy sinus was opened and found to contain an infected clot. Here the diagnosis was made by the ophthalmoscope alone, not only unsupported, but actually contradicted by the local signs.

16 July, 1907.

IV.

A CASE OF THROMBOSIS OF THE POSTERIOR CEREBRAL VEINS AND ARTERY, ENCEPHALITIS, PURULENT LEPTOMENINGITIS, LATERAL SINUS PHLEBITIS AND PARIETAL THROMBOSIS FOLLOWING MASTOIDITIS.

BY W. SOHIER BRYANT, A. M., M. D.,

NEW YORK.

A well-nourished woman 50 years of age was seen by the writer in consultation at the Manhattan State Hospital. She had a history of having had grippe more than four weeks previously, since which her mind had become unbalanced. The physical condition of the patient had been of negative interest except for diminished thoracic expansion, up to the day her temperature began to rise without any apparent cause. Three days previous to the rise of temperature her physical examination was negative. Her ears showed nothing abnormal.

On the fourth day after the commencement of the rise of temperature, rales and scattered patches of exaggerated breathing were noted at both bases behind. The right ear began to discharge thin pus. On the sixth day of the illness, the patient appeared very sick. Discharge from right ear continued. Some tenderness behind ear and indefinite earache. Lungs—dullness, rales, and exaggerated respiratory sounds over lower half of both lungs behind. Heart sounds very weak. On the seventh day the patient appeared better. Discharge from the right ear had ceased. No mastoid tenderness and little aural discomfort. Lungs slightly improved. Hemoglobin, 70 per cent. Leucocytes, 22,000. Ear irrigation continued.

On the eighth day the author did a complete mastoid operation for acute mastoiditis. Free myringotomy. The mastoid was opened in the usual way by first removing the tip, which appeared perfectly normal. The mastoid was very cellular, especially inferiorly and posteriorly, where the cells were filled with granulations, and there was free bleeding. Only two minims of pus were found which was located over the region

of the lateral sinus far back. The bone covering the sinus appeared healthy. The sinus was not exposed. The dura mater of the middle fossa was exposed through the tegmen antri posteriorly. It was very slightly congested and had slightly increased tension. The wound was washed and closed over a small drain. Iodoform gauze was put into the canal and a wet saline dressing outside. Time consumed, 35 minutes. Took anesthetic well. Returned to bed in fair condition. Examination of the pus from the cells showed large diplococci. Bacterial examination of the blood was negative.

The ninth day of the illness or first day after operation: Radial pulse very weak. Lungs apparently unaffected by the anesthetic. Leucocyte count, 20,600. Second day: Examination. Pupils small, regular and active. No stiffness of neck, Knee-jerk slight on right, absent on left. Achilles reflex present on left, absent on right. Some tendency to Babinski sign in left great toe. Spinal puncture, 5 cc. slow flowing, clear fluid withdrawn; no blood. Microscopic examination showed encapsulated diplococci similar to those found in the mastoid pus, staphylococci and a few bacilli and blood cells. No polynuclear leucocytes. Examination of the blood culture negative. Heart regular and rapid. Lungs unchanged. Patient dull, restless, no pain. No motor symptoms observable. Patient gradually became weaker, lungs more edematous. Radial pulse entirely lost. Temperature ranging from 105° to 106° followed by death on the eleventh day of illness and the third day after operation.

AUTOPSY BY DR. GLANVILLE Y. RUSK.

"Remains of a moderately well-nourished female. Pupils, circular, equal, moderately dilated. The amount of cerebrospinal fluid moderately increased. Over the right convexity, the subdural space shows a puriform cerebrospinal fluid and the convexity, especially along the Sylvian fissure, is covered with a somewhat granular fibrino-purulent exudate; the exudate is for the most part in the meshes of the pia-arachnoid or strips off with it, except near the anterior end of the temporal fossa, where for an area about 3×5 cm. the exudate is closely adherent to the dura. The veins over the right hemisphere are more prominent than over the left, the large veins over the temporal lobe and along the Sylvian fissure being the most marked. On opening the lateral sinus, it is found to contain an adherent

thrombus, starting anteriorly at the junction of the superior petrosal sinus with the lateral sinus and extending backward about 5 cm. along the superior and outer wall of the lateral sinus. A thrombotic plug is also present at the mouth of the jugular foramen. The point of most adherent pus formation to the dura lies about an area approximately 5 mm. in diameter, where the overlying bone has been removed during operation, and which is situated about 2 cm. above and slightly behind the auditory meatus. Smears from the intracranial pus show streptococci in short chains. Sections from the cortex show typical leptomeningitis with occasional streptococci, the inflammatory process not invading the brain substance. Unusually good examples of phagocytosis are present. The large nerve cells show examples of acute alterations of moderate grade."

Mastoid wound in good condition, clean and healing by first intention. The parietal thrombus of the lateral sinus covers and enters the mouths of the posterior cerebral veins. Thrombosis of right posterior cerebral veins and artery. Thrombus of jugular foramen appears to be more recent than the parietal thrombus of the lateral sinus. The meninges of the cerebellum and left hemisphere, normal. Ventricles, normal. Lungs filled with fluid. Death due to mastoiditis and pulmonary edema.

Capitulation.—The author was unable to find any references in literature to a similar extension of infection from the mastoid cells. The case seems to be unique in this respect that the infection from the mastoid crossed the lateral sinus without encroaching upon its lumen to any extent, entered the posterior cerebral veins and caused a fatal leptomeningitis which was out of all proportion to the comparatively slight mastoid involvement which was obscured by the concomitant pulmonary signs.

An interesting point shown at autopsy was the presence of two separate and distinct thrombi of different age, on the same side; one, of the lateral sinus; the other, of the jugular foramen.

The differential diagnosis between the complicated mastoid affection and lung affection was aided by the lack of parallelism of the respiration, pulse, and temperature curves and by the improvement in the condition of the lungs independent of the amelioration of the septic condition. In this case there was no macroscopic connection between the mastoid infection and the meningitis.

The temperature curve was not characteristic of sinus thrombosis since the rises were maintained for a time, blunting the characteristic saw-teeth. After the mastoid operation the question of jugular ligature was not raised because the temperature curve did not indicate thrombosis. The character of the old thrombus found at autopsy was not the kind likely to give rise to a characteristic temperature curve. The question of other intracranial explorations was considered and was decided against because of extreme weakness of the circulation. The autopsy showed the certain futility of any such procedure owing to the extensive leptomeningitis.

The history has been compiled from the hospital record kindly furnished by Dr. William Mabon, from the pathologic notes contributed by Dr. Glanville Y. Rusk, and from the author's own clinical and autopsy notes.

V.

A PLEA FOR CONSERVATISM IN THE TREATMENT OF CERTAIN AFFECTIONS OF THE NOSE AND EAR.

BY HARRY L. MYERS, M. D.,

NORFOLK, VA.

Some one has aptly said "it is better to be conservative in radicalism than radical in conservatism." The difficulty in this age of advanced surgery seems to be in finding the safe channel between the Scylla of conservatism and the Charybdis of radicalism. I fear that I shall not be able to point out the way in these troubled waters, but I hope to create a discussion at this meeting by which I may learn the better to avoid the dangerous rocks on either side.

With greater experience, I believe that there has arisen a spirit of conservatism among us which will continue to grow. The literature of the day confirms this and, recently, my courage to present this subject has been not a little strengthened by articles from some of our best men, expressing their belief in conservative methods of treatment in diseases of the ear and nose. It seems to me that it is in the surgical treatment of nasal diseases especially that more conservatism is needed. The antrum of Highmore, owing to its size, peculiar shape and dependent position, is not only particularly liable to infection but most frequently suffers from involvement of the sinuses situated above it, collecting their secretions which pour down, thus sometimes acting as a reservoir and assuming the appearance of guilt when it is really innocent.

The operation upon this sinus through the inferior meatus marks, in my opinion, one of the greatest advances in the surgery of the nose and is really, compared to the external operation, a victory for conservatism in that it is now used almost exclusively in work on this sinus, except in those cases in which there is sufficient necrosis to demand an external opening. I, however, think that even this operation, on account of its ease of performance and the lack of the necessity of general anesthesia in many cases, is apt to tempt us to destroy a

large piece of the inferior turbinate and a goodly portion of the inner wall of the sinus when frequently we could accomplish a cure by puncture through the inner wall of the sinus under the turbinate, and irrigation of the cavity every other day with a bland alkaline solution, over a period of three weeks or two months. Many cases, which have extended over months and whose discharges were decidedly offensive, have yielded to these mild measures at my hands. I do not mean to say that the cases I have been fortunate with were all cured by irrigation alone. When there is obstruction around the ostium of the sinus, whether it be from enlarged turbinate, deviated septum, polypi, or disease in the contiguous ethmoidal cells, I have removed these obstructions. In fact, I may say that in nearly all except acute cases, and in some of these, I think it will be necessary sooner or later to remove the anterior end of the middle turbinate. This is, however, a conservative measure and does not seem to do other than good. I am convinced that were more work done on the middle turbinate in these cases, and less on the inferior, results would in the end be better. It has been pretty well proven that the inferior turbinate has a well defined place in the moisture producing function of the nose, and I never could persuade myself that any more of it than absolutely necessary should be removed.

Before resorting to this operation we should be as certain as possible that the antrum is not acting simply as a reservoir, as under such circumstances we should fail to accomplish our end, and at the same time would produce an abnormality to no purpose. In most cases of chronic discharge from the antrum there will be found disease in other sinuses above it, and these sinuses should be treated and their secretion stopped, if possible, before more than irrigation is done to the antrum. Those cases of antral discharge where the trouble can be traced to diseased teeth, generally yield to drainage and irrigation through a generous opening in the tooth cavity, after the offending member has been withdrawn. Greater patience, however, is required in these cases, as time must be given for nature to throw off the necrotic tissue which may have formed in the floor of the antrum, around the diseased root. I may say here that I have experienced no trouble with food getting in through the opening in the alveolus, and I neither use an obturator nor a tube. I have experienced little trouble in the cavity closing, as I direct the patient to use a Moffett's dental

syringe which has a conical canula with a rather sharp point, thus being easy of introduction and serving to push back the parts effectively and without pain. Where there is much necrosis I believe the older operation from the outside is necessary.

We of the South have fewer cases of disease of the frontal sinuses with which to deal; most cases which have come under my observation have been rendered comfortable and in many cases cured of pain and discharge by conservative measures within the nose. I find that the removal of the anterior end of the middle turbinate and other obstructions to the infundibular opening, together with irrigation, has been of such service that few of my patients were willing to have resort to the external operation. I have never tried the method of boring into this sinus from the nose, and I never shall, as I consider it impossible for any one, however skillful, to know where the end of his drill is going, and as between this method of treatment and that of an external opening, the external method is the more conservative. I think, however, one may accomplish much by the cautious use of the curettes in enlarging the canal in an anterior direction, as recommended by Hurd; where there is bulging of the eye or tissues, or where pain is not relieved by conservative internal methods, the external operation often becomes a necessity.

I recognize that I am treading on dangerous grounds when I attack some of the methods used to-day in dealing with septal deviations, but I am afraid we are sometimes too bold in the use of the "window" resection operation. First, let me say that I feel that within proper limits it is the greatest advance in nasal surgery, and it will eventually be the principal method used for corrections of septal deformity. To my mind the danger of it lies not in its use, but its abuse. The more skillful we become in its technic, the more anxious we are to remove every bump and projection; in other words, we strive for a septum that looks perfect. In our enthusiasm for such a result I believe we not only at times remove more tissue than is necessary, but sometimes more than is safe. It seems to me that what we should strive for is to remove as little as possible in order to restore good nasal breathing regardless of appearances. We all know of very crooked septa where nasal breathing is perfect, and a projection which does not obstruct should be left alone. I am also of the opinion that we cannot yet afford to give up the nasal saw, for with it we can many times ac-

compish as good a result and we certainly feel more certain of the contour of the external nose.

The treatment of hypertrophic rhinitis has made advances in late years, due perhaps no little to an improved armamentarium and the established view that it rarely does harm to remove portions of the turbinate bodies with scissors and such instruments.

The careless use of the galvano-cautery has been so criticised that we are in a fair way to lose a very good means of relief in mild cases of this trouble; a means that is being used to-day by men of undoubted experience, and whose results are second to none. In those cases where there is no great thickening of the soft tissues but where the patient complains of great stuffiness at times, and in cases of intumescent rhinitis, where the patient either will not or cannot live an out of door life, I have found linear burns along the inferior turbinate with either the galvanic or chemical cautery to give me most excellent results, and I do not believe our enthusiasm for the newer cutting methods should lead us to abandon entirely the more conservative method in selected cases. Where there is great hypertrophy or deformity the snare or scissors have given me better results, but even then I think as little as possible of the bony turbinate should be sacrificed, except where the middle turbinate is concerned, and here I believe the cautery should never be used.

In chronic suppurative diseases of the middle ear, the time to relinquish conservative measures for radical treatment must necessarily depend upon several conditions. Environment, for instance, must be considered, for when a patient lives within easy reach of a competent aurist and has the means to obtain skilled treatment, he, in the absence of urgent symptoms, may frequently be made comfortable and rendered reasonably safe by the occasional use of intraaural methods of treatment, and I am of the opinion that we are scarcely justified in operating upon such a case radically when about the only symptom of annoyance is more or less discharge from the ear. On the other hand, the patient who is suffering from a chronic aural discharge with occasional acute exacerbation, who must live far removed from skillful treatment, had best submit to the radical operation.

Another condition which would indicate the necessity for radicalism is an unconquerable offensive discharge which in

some cases renders the life of the patient most miserable. When the clearing out of the tympanic cavity from the meatus either with the curette, chemicals, syringe or the operation of ossiculectomy (which, in my experience, while not always causing complete cure has never failed to improve) does not relieve pain and furnish good drainage and fails to greatly relieve all other symptoms, the radical operation should be considered.

When the probe demonstrates necrotic bone in the attic and when cholesteatomatous material is present in the discharges, we must sooner or later resort to radical measures.

In a report recorded some time ago by Dr. Harris before this society, he showed that during ten years in one of New York's institutions for treatment of ear diseases, out of 32,000 odd cases of ear diseases there were only 60 cases of intracranial involvement. In the same report he shows from statistics of many authorities that intracranial involvement was very rare.

Koerner made the statement that the radical operation is indicated as soon as a diagnosis of chronic purulent otitis media is positively made, but he also states that he has never seen or heard of a case of semi-mucopurulent discharge from the antrum, without obstruction to the outflow, that resulted in intracranial disease. In my own experience, extending over a period of thirteen years, I have seen meningitis once in a chronic case, serous meningitis once in an acute case, sinus thrombosis five times in acute cases, and twice in chronic cases. Since intracranial complications seem to be rare I have felt that we should employ every possible conservative means of relieving these cases before resorting to radical measures, which often not only fail to cure, but too often renders the patient's life more miserable.

VI.

PRIMARY NASAL DIPHTHERIA.

BY THOMAS HUBBARD, M. D.,

TOLEDO, OHIO.

Fibrinous rhinitis is a suggestive descriptive name for the disease under consideration, and like membranous rhinitis serves to link the old with the new pathology of diphtheria; but as membranous croup has given way to the more scientific expression, diphtheritic laryngitis, so will this type of rhinitis be called primary nasal diphtheria.

There is no doubt a form of membranous rhinitis, with exudate or exfoliation, resembling the true coagulation necrosis of diphtheria, caused by various bacteria, chiefly cocci, but these conditions are so rare that they may be eliminated from consideration.

Primary nasal diphtheria is a manifestation of the disease not generally understood by practitioners, and there is lack of clear forcible descriptions in some of the text books devoting many pages to diphtheria.

In passing I would make the criticism that we are less careful in the matter of diagnosis than in the early antitoxin period—that is, we do not utilize all of the aids to early accurate diagnosis. As the specific antitoxin has grown in favor we have become content to depend on the therapeutic test. It is all very well to say: "In case of doubt give antitoxin," but the trouble is that in laxity of diagnostic zeal we forget to doubt. I have seen primary nasal diphtheria overlooked so often by general practitioners that I feel justified in presenting clinical experiences and opinions of writers on the subject.

As compared with faucial diphtheria of the ordinary type, primary nasal diphtheria differs in two important features. It is nearly always a subacute or chronic disease process, developing insidiously, and the element of toxicity is comparatively

slight. These two clinical characteristics we should keep clearly in mind.

Diagnosis is often difficult, sometimes impossible, without the aid of the laboratory expert. False membrane may form in any part of the nasal fossae or nasopharynx, or we may have diphtheritic rhinitis with very little or no coagulation necrosis, only a hyperemia with a catarrhal flux. These are extenuating factors in tardy diagnosis, but even in obscure manifestations there are always suggestive clinical symptoms which should lead the careful observer to have made a laboratory diagnosis or make a timely therapeutic test by administering antitoxin.

Nasal diphtheria secondary to faucial diphtheria presents a different clinical picture. It is an extension of the acute disease process, with marked increase of toxemia, and rarely escapes diagnosis. It is mentioned only to exclude it from discussion.

There are two types of primary nasal diphtheria. In the one there is a formation of false membrane, sometimes to the extent of completely filling one or both nasal fossae with fibrinous casts, and a watery, acrid, mucopurulent discharge; and the other is merely a diphtheritic inflammation of the mucosa with a copious watery discharge of acid muco-pus. Altered blood pigment may give a reddish or brownish tinge to the secretion, and the nostrils and upper lip are usually excoriated. As a rule we find the fibrinous type in older children, and not rarely in young adults; and the catarrhal type is characteristic of primary nasal diphtheria in infants. In both, as previously stated, the disease runs a subacute or even chronic course, with very slight toxemia considering the area of tissue involved, and low mortality except in very young infants in whom mild sepsis and nasal obstruction interfere with nutrition. The disease as thus manifested in the nares is in a degree self limited but may run a course of several weeks, and there is a tendency to reinfection and relapse.

Extension from nares to fauces is rare and this is one of the puzzling clinical features. It is astonishing to see such an extensive area of infection with natural drainage into the throat and no manifestations of faucial diphtheria to be seen. Holt states that extension downward does take place, but usually in cases having the more acute type of primary nasal diphtheria. It rarely occurs in the chronic fibrinous type.

Another remarkable clinical feature of the chronic fibrinous type is that one naris may show evidences of extensive false

membrane deposit and the other be free from visible evidences of infection. The discharge may be from one nostril only, even in the advanced stage.

Culture tests show frequently pure Klebs-Loeffler, and while it is not proven that these are especially virulent, indeed I suspect from clinical experience that they are less than ordinarily virulent, yet the facilities for transmission are so much greater than in faucial diphtheria, the victim rarely being ill enough to be isolated or confined to one room, constantly using the family towel and drinking cups, and spreading the infection by contaminated fingers, that the net result is widely disseminated infection, and the most virulent type of faucial diphtheria may result in those infected.

This leads up to a more concise grouping of symptoms which should arouse suspicion. Simple acute coryza is the mask behind which primary nasal diphtheria invades the ranks of infancy and childhood. A watery, acrid, purulent discharge from one or both nostrils should always excite suspicion of primary nasal diphtheria, and if examination does not show false membrane the laboratory expert should be sent for and a culture test made. A foreign body produces similar symptoms but usually diagnosis is easy.

The systemic symptoms are, as stated, very mild and attract little attention. A degree or two of fever for a few days, malaise and anorexia, restlessness at night, due partly to nasal obstruction, are the principal symptoms. Toxemia is not very marked but there may be sequellae, paresis of groups of muscles as in ordinary faucial diphtheria. I have found albuminuria in several cases in which there were very slight systemic symptoms. There is often slight enlargement of cervical glands, not due necessarily to the diphtheric process but in part to mixed infection and local sepsis.

Primary nasal diphtheria in infants merits most careful study and it is often so obscure that it escapes detection even in institutions devoted to the care of infants.

I will quote from the address of Dr. John L. Morse (assistant professor of pediatrics, Harvard, assistant physician Children and Infants' Hospital):

"Membranous rhinitis is much more common in infancy than is usually supposed. It is generally of a very mild type and is usually not suspected until other children come down with pharyngeal or laryngeal diphtheria, and often not even then.

It is, perhaps, the most common source of the epidemics of diphtheria which develop in hospitals and institutions for infants and children. My experience at the Infants' Hospital has taught me to suspect every nasal discharge in an infant, especially if it is irritating.

"In spite of the greatest care in the admission of cases we found it impossible to avoid epidemics of diphtheria in the hospital from this cause until routine immunization was adopted. The babies now receive 500 units of antitoxin on admission and every three weeks afterward. Since this practice was begun, six years ago, but one case of pharyngeal diphtheria has developed in the patients in the hospital, although many unimmunized nurses and nursery maids have come down with it, and many cases have been discovered.

"The symptoms of nasal diphtheria in infancy are, as a rule, very mild. There is usually a little snuffles with a slight nasal discharge and a little nasal obstruction. There is no glandular enlargement and the constitutional symptoms are wanting or very mild. The nasal discharge, however, is often rather characteristic, being thin, watery and acrid, but, contrary to the usual statements, rarely bloody. It frequently irritates the upper lip, causing crusts about the nares and on the lip. A thin, watery, nasal discharge, which irritates the lip, should, in an infant, always be examined bacteriologically. The amount of the discharge varies markedly in different cases. There is almost never any membrane visible unless the speculum is used, and even then it may not be seen. The diphtheritic process practically never spreads backwards and downwards to the pharynx and larynx. The diagnosis must depend, therefore, in most cases, on the bacteriologic examination of the nasal discharge."

One of my earliest cases was in a family of seven children. The five-year-old boy had diphtheritic laryngitis, dyspnea so extreme that he was unconscious. I intubated and respiration ceased altogether. Tracheotomy and artificial respiration for 15 minutes restored consciousness. I then looked over the family and found the culprit. A lad of twelve had an excoriated upper lip from an irritating watery, purulent discharge from both nares. A small deposit of false membrane was seen on the septum. He had been in this condition for more than ten days, and without the slightest throat symptom or effect on his general health, and had been daily in school.

Administered antitoxin to all of the family. Within 24 hours three children had faucial diphtheria and two more had diphtheria of larynx, but large doses of antitoxin saved them from intubation. All recovered. The discharge from the primary nasal diphtheria case showed Klebs-Loeffler bacilli.

In one experience I had to combat the opinion of the family physician, a man of wide experience, as he was opposed to the administration of antitoxin for what he termed purulent catarrh. The lad was apparently in good health although there had been a watery discharge from both nostrils for more than a week.

He came to me on account of nasal obstruction, and I discovered large plaques of firm white membrane on both sides of the septum. In this case I administered 6000 units of antitoxin and soon had confirmation of diagnosis by report of bacteriologist. Within 48 hours a younger brother and sister had severe faucial diphtheria and soon after the mother and an aunt had the disease. A baby of 12 months was given an immunizing dose and had only catarrhal symptoms, possibly mild nasal diphtheria.

Five or six cases of primary nasal diphtheria have been brought to my office and nearly always the diagnosis "foreign body in the nose" has been suggested by the family physician. In fact, two had undergone operation for removal under general anesthesia. This error is referred to by Northrup (Northnagel's System of Medicine) as being frequently observed in dispensary service.

Frequently primary nasal diphtheria runs its course undiagnosed; and a tell-tale sequela, in the nature of paresis of certain muscles, reveals the true nature of what was considered coryza. A young man of 17 returned from boarding school in apparently good health to spend a vacation. It was noticed that the voice had a peculiar nasal character, and he swallowed fluids with difficulty. Examination showed one-sided paresis of the velum palati, and an excoriated septal mucosa, posterior third, with small plaques of false membrane. Bacteriologic examination showed Klebs-Loeffler bacilli. This was about three weeks after he had apparently recovered from what was termed coryza by the dormitory physician, although at the same time one boy was sent home with tonsillitis.

This case was unusual in that he did not have the characteristic acrid nasal discharge, the disease being limited to an area

involving the posterior third of septum on one side only, and it was of decidedly chronic character. No antitoxin was administered and the throat paresis gradually subsided.

In another similar case, a child of three, the diagnosis was not very positive, no bacteriologic examination having been made, but the therapeutic test was decidedly confirmatory. Paresis of velum developed in two weeks. In these two cases, and in others, I have been impressed with the lack of virulence of the infection, as evidenced by the chronic course of the disease, and by the fact that many other children in close contact with the one sick did not contract the disease.

Concerning the degree of virulency of the bacilli in these cases of the chronic type, Dr. Thomas B. Cooley of Detroit makes the observation, based on an extensive experience in the contagious disease wards of the Boston City Hospital, that the presence of the copious sero-mucus discharge probably has an inhibitory influence on the growth of bacilli, and thus effects the virulency, but one cannot draw any positive conclusions. A given case of very mild chronic type may produce an acute fulminating type of faucial diphtheria in another individual. Dr. Cooley confirms the opinions of other writers, that primary nasal diphtheria, from the fact that it is so often overlooked, is the most potent factor in the causation of epidemics. In his experience it is very difficult to find false membrane in infants and he would depend more upon the character of the nasal discharge. Contrary to the opinion expressed by Morse, Dr. Cooley has often found altered blood pigment in the discharge from the nares, and especially in infants.

The treatment is essentially the same as that of faucial diphtheria, except that all writers are agreed that in typical primary nasal diphtheria relatively smaller doses of antitoxin seem efficacious. It is astonishing how quickly all symptoms melt away after a dose of 3000 units. But yet, considering the fact that diagnosis is made late, as a rule, I am in favor of a rather large primary dose, 6000 units in first 24 hours.

The temptation is to attack the local condition with antiseptic irrigation or douching, but all are agreed that it is futile and not without danger to accessory sinuses or the ears. A mild detergent solution dropped into a nares with a medicine dropper is sufficient.

It should always be borne in mind that these cases of primary nasal diphtheria harbor bacilli longer than the ordinary faucial type, and the period of isolation should be prolonged.

VII.

A PLEA FOR LOCAL ANESTHESIA IN OPERATIONS ON THE MAXILLARY SINUS.*

BY GORDON KING, M. D.,

NEW ORLEANS.

In resorting to surgical procedures for the relief of conditions which, in themselves, do not entail risk to the patient's life or interfere so seriously with comfort as to render life burdensome, it becomes the prime duty of the surgeon to minimize in every way possible the risk to life incurred by the operation itself consistent with analgesia, avoidance of shock and thoroughness of execution.

How doubly unfortunate does it seem when a fatality occurs from the use of a general anesthetic in the performance of some simple operation for a minor ailment, for which, if operation is essential for relief, a local anesthetic skillfully and judiciously employed would have been sufficient to prevent pain, and at the same time would have been comparatively free from danger to life. Few of us but can recall to mind in the sphere of his observation or experience some catastrophe of this kind for which vain regrets offer but poor consolation.

Recognizing as we must, the ever present danger in any general anesthetic and the impossibility always to foresee and to prevent a possible accident, it is but just that every patient whom we are to subject to this danger, or those responsible for his welfare, should be made aware of it, however slight the risk may appear to be. Among the more intelligent we take it for granted that the risk is understood and accepted without discussion, but often the surgeon is anxiously interrogated as to any possible danger to be incurred, and in such instances it has always been my custom to express my opinion frankly, although regretting the undue anxiety this may cause. For these reasons and others of practical bearing, I have within the past few years endeavored to restrict as much as was practicable my use of general anesthetics and resorted more to local.

*Read before the twenty-ninth annual meeting of the American Laryngological Association, Washington, D. C., May, 1907.

I have proved to my own satisfaction that in many cases in which formerly either through precedent or habit I relied upon general anesthesia, I now can accomplish equally good results under local anesthesia.

Especially is this true of operations about the mouth and upper air passages, where in addition to the danger from the anesthetic we have to contend with the annoyance and delay incident to nausea and vomiting, the inhalation of blood and secretions into the larynx, and the frequent interference with the administration of the anesthetic. These annoyances so often hindered me in antrum operations, where more than half the time is occupied with the administration of the anesthetic and delay caused by retching and the accumulation of blood and mucus in the throat, that I decided some time ago to try the efficacy of local anesthesia. The result has been even more satisfactory than I anticipated, and for two years past I have used a local anesthetic exclusively in these operations.

The antral region is peculiarly well adapted to local anesthesia, and even the sting of the hypodermic needle can be obviated by a preliminary swabbing of the mucous membrane in the canine fossa with a ten per cent solution of cocain.

Being still a loyal advocate of the Caldwell-Luc operation for antral empyema rebellious to the less radical treatment, I shall describe briefly the manner in which this operation can be performed practically without pain and in less than half the time required when a general anesthetic is employed. Including the first few minutes expended in benumbing the mucous membrane of the canine fossa by topical application, the entire procedure has on an average taken not more than seventeen to twenty minutes.

I begin by swabbing the mucous membrane briskly with a ten per cent cocain solution, then when the part is insensible to the prick of the needle a solution of cocain (2 per cent) and adrenalin chlorid (1-2000) is injected along the line of incision, at first superficially into the sub-mucous tissue and then more deeply into or under the periosteum. This done, the incision is made down to the bone and the periosteum elevated until the exact situation of the infraorbital foramen can be made out, when to insure more perfect and widespread anesthesia a few drops of the solution are injected into or around the trunk of the infraorbital nerve at its point of exit.

VIII.

CONTRIBUTION TO THE STUDY OF TUBERCULOSIS OF THE NASAL MUCOSA—AN ETIOLOGIC AND CLINICAL STUDY.*

BY HENRI CABOCHÉ.

TRANSLATED BY ALBERT MILLER, A. B., M. D.

Clinical opportunities having given me occasion to see several cases of lupus and so-called tuberculous tumors of the nasal mucosa, I was soon persuaded that the classic scheme of tuberculosis of the pituitary membrane did not probably correspond with the facts, and I conceived the idea of reviewing the works upon which it is based.

In looking over the different works one is struck by the varying classifications of these writers.

Cartaz in his epoch-making memoir¹ and Boutard in his thesis² described two forms of nasal tuberculosis: one, usually primary, resembling a tumor; the other, usually secondary, taking the form of a tuberculous ulcer, clinically resembling other ulcerations of the mucosa. Further, ulcer and tumor may coincide.

This is the description which we again find in our classics (Lermoyez, Garel, Moure).

Plicque³ describes tuberculous granulation, ulceration and tumor. The granulation, which is rare, is gray granulation which later gives rise to ulceration.

Olympitis⁴ in his thesis inspired by Tissier, distinguishes no less than eight forms of pituitary tuberculosis which he divides into: Acute miliary, polypoid, infiltrated, lupus, tubercular ozena, submucous abscess, ulcerous, caseous. The last two as well as the acute miliary form are secondary to pulmonary localization, the others are primary.

Koschier⁵ in a very complete and interesting memoir gives twenty-one personal observations of nasal tuberculosis. He distinguishes an ulcerative form, a nodular or neoplastic form, and an osseous and cartilaginous form, histologically and clinically analogous to tuberculosis of other bones.

*From the *Annales des maladies de l'oreille*, etc., Oct., 1907.

Chiari (Affections of the Nose, translation by C. Breyer, 1905) distinguishes a chronic catarrhal form resembling ozena, an ulcerous form, and a neoplastic form.

All these writers distinguish tuberculosis, properly so-called, from lupus of the pituitary membrane, but they do not make clear how to determine clinically what is lupus and what is tuberculosis.

Cartaz, Boutard, Pistre⁶ include neoplastic formations in tuberculosis; Raulin,⁶ on the contrary, places them in the lupus class.

Similarly, in Germany, Schoeffer considers his six cases of tumors as the clinical manifestation of nasal tuberculosis, on the grounds that they were not accompanied by any lupic cutaneous or mucous manifestation. Bresgen, on the other hand, objects with reason I think, that cutaneous manifestations may sometimes be very tardy, appearing perhaps two years after the beginning of pituitary lupus, and for these reasons considers Schoeffer's growths as lupus tumors.

Olympitis, while placing Schoeffer's tumors in the tuberculous class, recognizes that they constitute a transition form between tuberculosis and lupus. Likewise his infiltrated form, "limited to lupus." Now, there is not only a relation between this form and lupus, but, as we shall see, an absolute identity.

Hinsberg⁸ in a work on the relation of ocular affections to the nasal mucosa, reports four observations of pituitary tuberculosis and one of lupus.

I have soberly asked myself, upon reading this otherwise very interesting note, upon what characters the author depends to declare that one case is tuberculosis and another lupus.

Here, very much abbreviated, are two of his observations of nasal "tuberculosis:"

1. Young girl of 15. The right inferior meatus is filled with a granular mass, in places ulcerated, coming apparently from the inferior turbinate; on the left side a similar tumor but much smaller. In the pharynx, the right tonsil, the pillars and neighboring parts of the velum are infiltrated, thickened and pale. The surface is studded with small nodules but without ulceration; this condition extends to the epiglottis which is thickened, immobile and covered with similar nodules. Later on, there appeared nodules on the mucosa covering the arytenoids.

2. Girl of 17; came to the clinic because of an eruption on the lobule of the nose and left lower eyelid. There was found at the right nasal opening a severe eczematous condition with crusts; the skin at the summit of the nose was reddened, with some nodules. The right side of the septum, at its lower anterior portion, shows a mass of granulations covered with crusts. The right inferior turbinate is similarly affected, and the left is covered with crusts and granulations. In the nasopharynx, right side, there are some nodules; some nodules also on the skin of the left lower eyelid.

Here now is the case labeled lupus:

Man of 48, previously treated at the dermatologic clinic for cutaneous lupus of the nose. At the time of examination the nasal fossae show, besides a deviation of the septum, numerous small nodular tumors on the mucosa of the left inferior turbinate and both sides of septum. Three years afterward there was seen the same turbinate lesions with an added perforation of the cartilage.

Evidently in these three cases there was a single disease.

Massei, in his work published in the *Revue hebdomadaire de Laryngologie*,⁹ on nasal tuberculosis, declares the division into lupus and tuberculosis to be superannuated, and proposes a complete erasure of the term lupus from the medical vocabulary.

Escat, on the other hand,¹⁰ has never had occasion to see any other manifestation of tuberculosis in the nasal mucosa than lupus.

It is thus to be seen that the confusion is complete, and, as I said at the outset, the question of nasal tuberculosis is to be taken up anew. I believe that it will be of interest to examine the accepted classic forms of nasal tuberculosis, and I hope to be able to demonstrate that they may be reduced to a sole and single modality—lupus.

I will therefore take up successively the following questions:

1. Is there an ozenous form of nasal tuberculosis?
2. Is there ground for recognizing an osteo-cartilaginous form of nasal tuberculosis?
3. Is there ground for making a distinction between tuberculosis and lupus of the pituitary membrane.

After solving these questions, I will discuss the clinical aspect of nasal tuberculosis, its etiology, its progress, its complications, its diagnosis and its treatment.

I.—IS THERE AN OZENOUS FORM OF NASAL TUBERCULOSIS?

RELATION OF OZENA AND TUBERCULOSIS
OF THE NASAL MUCOSA.

Let us first examine the observations upon which this clinical form has been established. These cases, now ancient, are three in number, and I will give them in extenso. One belongs to Hajek, the other two to Demme.

(a) Hajek (in These Boutard). Tuberculous ulceration with ozena in a youth of 13.

Youth, aged 13, nose flattened, exhaling a very bad nasal odor. The interior of the nose was filled with crusts, the bony septum was partially destroyed, and there were pustular granulations in the bottom of the ulcers. The mucosa of the middle and lower turbinates on both sides was dry, thinned and greatly atrophied. The patient had been in this condition for a year and a half. For a year there had been considerable swelling of the cervical glands, which had improved under iodized baths without any change in the nose.

A minute examination did not show any symptoms of hereditary syphilis; his parents having died long before, no information in this respect could be obtained. For diagnostic purposes I removed a piece the size of a pea from the edge of the septal ulcer. I made a section and found in a spot rich with cells, characteristic tubercles, among which some had at their center giant cells with their nuclei and a few bacilli. Others appeared to be undergoing caseous degeneration. Application of lactic acid to the ulceration. His condition improved temporarily, but the ulceration finally increased progressively and the malady followed its usual course.

(b) Demme.¹¹ (Berl. klin. Woch., 1883, Bd. xx, No. 15):

In a particularly grave case of rhinitis, which presented itself as an infantile scrofulous ozena, the writer observed in a 12 months old boy little groups of grayish yellow nodules showing up clearly on the nasal septum after cleaning out the crusts which obstructed the nose. These nests were irregular and had small ulcerations on their surface. The bottom of the ulcerations was filled with a yellow sanious mass covered with pus. In this case hereditary syphilis was determined with certainty. However, it turned out to be an ulceration of the mu-

cosa, of a tuberculous nature. In fact, the child was seen a few months afterward, and he then had pulmonary phthisis."

(c) Demme (id.).

February 28, 1885, there was brought to me a little boy, 8 months old, Fritz B., who had ozena. A complete physical examination of the child led to the exclusion of hereditary syphilis. The antecedents were in accord with this view, and the diagnosis of hereditary tuberculosis was made. It was then learned that the child had been taken into a family in which the father had acute miliary tuberculosis. The ozena appeared in the baby at about his sixth month, two months after his residence with this family. On the right septal mucosa was seen a group of small nodules identical with those above described. Some of these were the seat of lenticular ulcerations of unequal depth and with crenated borders. In five preparations from the nasal discharge made February 28, only one contained tubercle bacilli. March 1, after a nasal irrigation with warm water, the patient had a violent attack of sneezing, followed by an abundant nasal secretion which when examined under the microscope after staining with fuchsin, revealed a considerable number of bacilli, almost as great as the number found in acute phthisis. March 2, there were convulsions with high fever; another bacteriologic examination with the same result. With the symptoms of an acute meningitis of rapid onset the patient died March 5, after frequently repeated general convulsions and contracture of the lower extremities.

At autopsy there was found a tuberculous meningitis of the base of the brain. Numerous miliary tubercles were along the branches of the Sylvian vein as well as the capillaries of the inferior surface and the frontal lobes, notably the peripheral cerebral veins, anterior and inferior. No tubercular products in the brain or of the cord. The lungs, bronchial glands and abdominal organs were exempt from tuberculosis. The mesenteric glands appeared swollen and soft. No caseous foci were found. In the axillary folds were some isolated hypertrophied glands; no caseation on section.

The ulcerous masses in the nose, when microscopically examined, showed bacilli in moderate numbers, either in isolated groups or included within giant cells.

In some sections from the miliary tubercles of the pia mater a very few bacilli were found. The author concluded

that it was a case of direct tuberculosis infection of the nasal mucosa, coming directly from the foster father, who had pulmonary tuberculosis. The tubercular infection of the nasal mucosa was carried by the vascular route, and thus implanted in the pia mater and base of the brain.

As may be seen, these observations have dealt with a special form of ozena, and all three mention ulcerations of the mucosa, although everybody nowadays agrees upon the absence of ulceration in true atrophic rhinitis.

The ulceration mentioned in Hajek's case was of bone. Now, in none of my personal observations of nasal tuberculosis nor in those which I have collected from various publications (covering a century) have I found this species of ulceration. It may be said then that perforation of the bony septum in nasal tuberculosis is a rarity.

On the contrary, it is a classic feature of tertiary syphilis of the nasal fossae which may be accompanied at an advanced period of evolution by atrophy of the inferior turbinates. So that one may ask if, in this case, there was not a tubercular infection engrafted upon a nasal syphilis. To be sure, the observation states expressly that there were no stigmata of hereditary syphilis, and the great authority of Hajek should be a guarantee of the fact. However, specific treatment does not appear to have been tried. This is plainly a valuable item which is missing.

The observations of Demmè have dealt with an extremely rapid tuberculosis. In the case where a bacteriologic examination of the secretions was made, numerous bacilli were found, "almost equal in quantity to that found in acute phthisis." Autopsy showed numerous miliary tubercles along the branches of the Sylvian artery and vein. One gets the impression that it was a rapid, massive, recent infection, which might have been subsequent to the appearance of the ozena.

So, one might ask himself if, in these cases, there was not a simple coincidence, or rather a juxtaposition of acute tuberculous infection and true ozena. Hence these observations do not seem to me beyond criticism.

I may add that the rarity of these cases contrasts singularly with the considerable number of cases of pituitary tuberculosis not accompanied by ozena. Is it not astonishing if Koch's bacillus has ozenogenous power, not to see it oftener in action?

However, these cases ought to be taken into consideration. They show, indeed, one thing: that Koch's bacillus may live in good comradeship with the bacilli of ozena.

Clinical researches which I made long ago have convinced me that atrophic rhinitis is only a larval form of nasal tuberculosis; (Soc. Française de Laryngologie, June, 1907.) But Koch's bacillus does not produce ozena, the bad odor; it causes the atrophy, the sclerosis of the turbinates, just as it elsewhere produces renal sclerosis or periportal sclerosis, and this atrophy through the new condition which it provokes in the nasal fossa (drying of secretion, etc.), would favor secondary infection by the microorganisms of ozena.

Thus understood, tuberculous ozena would be relatively frequent. Its existence is not yet demonstrated. Further researches are necessary.

II.—IS THERE GROUND FOR RECOGNIZING AN OSTEO-CARTILAGINOUS FORM OF NASAL TUBERCULOSIS?

Osseous manifestations of nasal tuberculosis are extremely rare; I know of but two cases, which I shall recount:

Case I (Verneuil-Ruault in These Boutard). In 1887 Verneuil sent to Ruault a woman of 40 whom he had treated two years for a nasal affection which began as a tumor in the floor of the right nasal fossa. Verneuil did an ablation, and an examination by Nepveu showed tuberculosis.

A cure could not be obtained. The tumor did not recur, but the wound ulcerated and the cartilaginous septum was soon destroyed. Weekly cauterizations with the thermocautery for two years, when the patient was sent to Ruault.

The latter found a grayish ulcer with jagged edges occupying over a third of the floor in the right nasal fossa and a smaller area in the left; no trace of the cartilaginous septum except at its upper part. The vomer was carious in its whole anterior portion. At the opening of the right naris there were some small tubercles on the skin, of lupus appearance. For several weeks the voice had been roughened and deglutition painful.

Pulmonary and laryngeal lesions carried off the patient.

Case II (Koschier, Case XV in the work already cited). Woman, H. T., 22 years, admitted May 17, 1894. For a year had noticed that air did not pass through the nose.

Externally the nose presented on both sides a very large bridge, larger than normal. The skin was inflamed and red, especially on the bridge. Palpation of the nose caused lively pain. The wings of the nose were swollen somewhat, but of natural consistence. Both nasal fossae were filled with a large tumor causing complete impermeability of the nose, a soft, nonulcerated tumor seated on the septum in front and completely hiding the septum laterally and posteriorly. Nothing abnormal by posterior rhinoscopy. Throat, larynx and lungs normal; no bacilli in sputum in spite of repeated examinations. Examination of a portion of the tumor showed tuberculosis.

Under anesthesia the greater part of the tumor was removed with a bistoury; it was then found that the lateral parts of the nasal fossa, in particular the inferior turbinate and floor, were greatly infiltrated and showed ulcerations in several places. With the probe denuded bone was felt in several spots. Operation was then stopped; tamponment with iodoform gauze; subsequent dressing; application of lactic acid and insufflation of iodol. Death July 3, 1895. At autopsy, acute basilar tubercular meningitis with acute internal hydrocephalus and cerebral edema. Chronic tubercular periostitis and perichondritis of the nose. Tubercular otitis media and caries of the mastoid process. Chronic tuberculosis of both pulmonary apices.

Authors who have occupied themselves with nasal tuberculosis do not fail, for the most part, to quote the opinion of Volkmann upon primary caseous osteomyelitis of the nasal bones, invading the nasal mucosa secondarily, which he regards as a frequent cause of *ozena*. Now, when this work is consulted, it is not a little surprising to find that it deals with the character and significance of fungous arteritis. In one passage in the work, Volkmann voices the opinion above mentioned, but without the support of any clinical facts, much less any histologic examinations.

As for the cases above given, I find them extremely debatable.

Nothing proves that the bone lesions in the first case were tubercular. I even believe that weekly treatments with the thermocautery for two years were amply sufficient to provoke the osteitis of the vomer.

As for the second case, I hope not to be taxed with exag-

geration in saying that if this observation had been presented to an unprejudiced reader, omitting the bacteriologic examinations, he would have diagnosticated tertiary nasal syphilis with osteo-periostitis of the nasal bones proper.

There is nothing lacking. Thickening of the nasal dorsum, with redness, edema, and lively pain upon pressure, and smooth swelling from infiltration of the inferior turbinate which the probe found to be denuded and carious at the bottom of an ulceration.

Trial with specific treatment is not mentioned, and it may be asked if it was not a mixed lesion, syphilitic and tubercular. The fact is not impossible.

At the Vienna Laryngologic Society, November 9, 1904, Dr. Marschik¹⁴ reported a case of tuberculosis of the nose and velum resembling syphilis, and improved by mercurial treatment. In the same session, Dr. Heindle¹⁵ stated that he had seen analogous cases where there was no trace of syphilis in the history, in which examination demonstrated the existence of tubercles and which healed under antisyphilitic treatment.

Hence, these cases are not convincing as to the solely tubercular nature of the bone lesions observed.

In any event they do not suffice to justify the existence of an osteo-cartilaginous form of nasal tuberculosis. I do not mean to deny the possibility of tuberculosis of the nasal bones; here, as in other bones, Koch's bacillus could propagate, but that is a thing altogether different from what we are concerned with in this paper. And I do not believe that the question of nasal tuberculosis is clarified by including tubercular osteitis of the maxilla, frontal or ethmoid.

It will be seen in the chapter on pathogenesis that I completely reject, on clinical grounds, the osseous origin of common nasal tuberculosis.

III.—IS THERE REASON TO MAKE A DISTINCTION BETWEEN PITUITARY TUBERCULOSIS AND LUPUS?

We have seen, at the beginning of this paper, how much confusion reigns in authors' descriptions, and what one calls common tuberculosis another calls lupus.

To solve the question it will then be necessary to set forth as precisely as possible the characteristics of pituitary lupus,

which have been only vaguely described in classic treatises. These characteristics being known, we can review the cases reported as nasal tuberculosis, and see if the lesions therein described differ or not from those considered to be lupus.

I will first take up the characteristics of lupus and discuss the cases afterward.

A.—Objective Signs of Pituitary Lupus.

The objective symptoms which I am about to describe are those which I observed in twenty patients who were attacked simultaneously with lupus of the nose or face and with pituitary lesions, most of whom were sent to me by Dr. Balzer. Four only had intranasal lesions alone, in every respect similar to those observed in the ones who had cutaneous lupus.

In spite of the apparent identity, I subjected two of these patients (Cases IV and XV) to mixed antisyphilitic treatment. The treatment was absolutely without effect upon the lesions. To make it stronger, I inoculated two guinea-pigs with a bit of mucosa taken from each of these patients; both inoculations were positive.

The failure of specific treatment and the positive inoculations satisfied me of the tubercular nature of the lesions. The coincidence of cutaneous lupus showed, in another way, the true significance of these lesions.

It follows from fifteen cases published herewith further on (to which can be added six others published in the *Annals of September, 1906*¹⁶ and of *January, 1906*)* pituitary lupus may assume four different forms or aspects:

Nodular lupus,
Vegetating lupus,
Lupus tumor.
Ulcerous lupus.

1. *Nodular lupus*.—It is characterized by the appearance on the pituitary mucosa of nodules, usually of a pale rose color. The number varies according to the time when seen; there may be only one or two at the beginning. They are two or three times the size of a pin-head. They lack the lustre of normal mucosa, being dull and roughened. Soft upon con-

*The two latter cases I published as naso-lacrymal tuberculosis. They were, in fact, lupus, for the patient in the second case, Louise B., had a double facial lupus, and I have recently seen the little patient in Case 1, who showed typical pituitary lupus.

tact with the probe, they are separated from each other by small irregular grooves, some ulcerated, others cicatricial.

When they are numerous they infiltrate the mucosa in a series of nipple-like lobules separated from each other by the grooves above mentioned—this is the mammillated infiltration characteristic of pituitary lupus. At this stage the mucosa is soft to the probe and does not contract under the influence of vaso-constrictor medicaments.

The more or less abundant crusts often mask lesions to which they slightly adhere. The lesion may be seated in any part of the nasal fossae, but it always begins anteriorly, as we shall see in the chapter on the course of the disease; for the moment I want only to give the characteristics of lupus in a fashion such that they may be compared with those in case reports which, rarely it is true, have dealt with lesions at their commencement. The mammilla with grooves surrounding it is, in a way, the elementary lesion of pituitary lupus—it is always found in some form.

2. *Vegetating Lupus*.—It is characterized by a more or less extensive hyperplasia of the mucosa—a pale rose or bluish lilac hyperplasia with a series of crests separated by little ditches.

I cannot make a better comparison of the appearance given by the mucosa than to the geographic wall charts in relief designed for primary classes. One sees minute hillocks separated by miniature valleys. Each of the hillocks, sometimes rounded like a dome, sometimes terminating in a sharp crest, is made up (the same as the valleys) of an agglomeration of very small nodules the size of a pin-head, separated by extremely narrow grooves, giving to the lesion as a whole a muriform aspect which is peculiar to it.

These lesions always feel soft to the exploring probe. Especially upon curetting is this extreme softness perceptible. The softness is even a good means of diagnosis when, in the course of treating lupus, one is uncertain whether a given region is or is not lupic.

The group of lesions occupies a more or less considerable surface, according to the period when observed; at the beginning it may occupy solely the anterior zones of the nasal fossae (Case IV), or a large part of the septal mucosa (Case III); later it may involve almost all the nasal mucosa (Case VI).

3. *Lupus Tumor*.—This merits description apart from the

vegetating form. Pedunculated or sessile, of variable size (that of a hazelnut, a walnut, the entire nasal fossa in one case) the tumor is of a pale bluish white color, sometimes red (probably in consequence of secondary infection). Of smooth appearance on superficial examination, one sees on its surface (personal case) a noteworthy number of grayish yellow elevations the size of a millet seed. It is elastic and bleeds little or not at contact with the probe.

It may be almost anywhere in the nasal fossa; on the septum, floor, inferior turbinate, middle turbinate (anterior or posterior), as may be verified by reading my cases.

Finally, it is almost always accompanied by some other manifestation of pituitary lupus (nodules, vegetations, perforation of the cartilaginous septum). I may add that it may be single or accompanied by other identical manifestations; we then have a form intermediate between the vegetating form and tumor.

4. *Ulcerating Lupus*.—Ulceration is an eventuality which may ensue in the course of the preceding varieties. It is an ulceration with irregular borders, sometimes polycyclic, with granular bottom, sometimes of a necrotic appearance. Resulting from the breaking down of preexisting lupic tissue, it is always surrounded by a zone of mucosa showing one of the lesions above described. Most often it occupies the cartilaginous septum, but it may be seated upon the subseptum, the floor, or the inferior turbinate.

If it comes on in the course of a vegetating lupus, it is always found to be elevated above the healthy mucosa; it thus gives the geographic relief chart appearance, which I have mentioned. It only attacks the cartilages late.

If it develops upon a nodular lupus, it easily causes perforation of the septum, which is the more easily produced in bilateral lesions of the septum. It is probable that the loss of circulation in the cartilage from destruction of its nutrient mucosa, brings on the necrosis.

The perforation presents peculiar characters: It is rounded and regular. The dimensions vary from a pea to a two franc piece. The border is surrounded with a collarette of fungosities, soft, bleeding, pale or red, more or less voluminous.

It is not rare—a somewhat characteristic fact—to find a part of the periphery cicatricized and showing a thin border,

while the rest, in full swing, is thick, tripled in volume and fungous.

A capital fact: it attacks exclusively the cartilaginous septum; I have never seen lupus accompanied by a bone lesion.

Perforation of the cartilage is an extremely frequent manifestation of lupus at a certain period of its evolution, hence it has high diagnostic value. Its presence, accompanied by other lesions difficult to interpret, not involving the bone, is sufficient, I hold, to give these lesions a lupic significance.

Ulceration, speaking precisely, is less a form than an end-stage of lupus.

I would say the same of the stenosing form, which Raulin describes in his thesis. This is an atresia of the naris, encroaching more or less upon the nasal fossa, and simply means the cure of an old lupus of the vestibule and anterior nasal fossa. It is strongly indicative of lupus.

Sometimes it is characterized by the formation near the posterior narinal border of a vertical and frontal thickening with an upper concave edge, three or four millimeters high, barring in a fashion access to the nasal floor. I have seen this in one case; it has also been pointed out by Mygind⁸⁷.

In other cases there is actually produced a fibrous tunnel through which the parts constituting the nasal fossa are seen with difficulty (Raulin and Cases X, XI, etc.).

I wish finally to call attention to three frequent phenomena in the course of pituitary lupus, and consequently of great significance if we should find them mentioned in case reports. They are: Destruction of the alae nasi, lymphangitis of the lobule and "eczema" of the nares.

The nares may be more or less destroyed, eaten away by the breaking down of lupus cutaneous nodules. Various deformities result, the least of which is more or less reduction of the narinal dimensions with characteristic vermicular cutaneous cicatrices, or if extensive, the horrible disfiguration called the "death's head." This is not the destruction which I have in view, but the progressive destruction of the alae nasi, unilateral or bilateral, without any nodules in this region. I have noticed it several times, especially in patient of Case I. The alae nasi were the seat of a smooth scar tissue production which led to their progressive wasting; during the five years that I cared for this patient I have seen them become an arch, the concavity below, thus gradually uncovering the septum. In an-

other case I have seen almost total destruction of the left ala, exactly resembling cases of rhinocosis. There may have been a tuberculous trophic process in this case, or in the first, a sclerodermic tuberculosis, analogous to that recently described by Milian¹⁷.

I consider the symptoms in both cases sufficiently characteristic of lupus. Lymphangitis of the lobule is very frequent in the course of pituitary lupus. It gives to the extremity of the nose, a swollen, red aspect, which I have often noted, sometimes long before the appearance of the cutaneous nodules, at times even the initial symptom at the beginning of pituitary lupus (I shall recur to this fact). Is it a tuberculous manifestation or simply a transitory infection of the skin with other microorganisms? I cannot say; it is frequent, may be slight or intermittent, and seems to me to have much significance in being always associated with pituitary lupus.

I would say as much for eczema of the nares, which I have frequently found mentioned in observations of pituitary tuberculosis. This eczema may well be a tuberculous manifestation.

Hutchinson (cited by Lenglet, *Pratique Dermatologique*, p. 323) admits that eczema may precede the evolution of lupus, but he remarks that some cases of lupus preserve, during their whole course, the aspect of prelupic eczemas. This is also the opinion of Jodassohn (id.), who regards certain chronic eczemas of the nose and upper lip as the preliminary stage of lupus. Tuberculin often gives a positive reaction in these cases. Also, we must distrust certain varieties of so-called chronic eczema which transform themselves into lupus. Certain prelupic eruptions are tuberculous, and some eczemas are doubtless of the same origin and merit the appellation of tuberculous eczemas, or rather prelupic eczematiform dermatitis (Lenglet, loc. cit.).

When we recall that the progress of lupus is extremely slow; that it lasts for years; that recurrences are extremely frequent in spite of treatment methodically applied, we possess those principles which permit us to criticise case reports.

B.—Criticism of Case Reports.

From the above exposition, based upon my twenty observations, I believe I have a right to draw the following conclusions:

1. We can surely list as pituitary lupus the cases of tubercu-

losis where there was remarked one or several of the following signs:

Mammillated infiltration of the mucosa, which I regard as the typical lesion of lupus.

Perforation of the septal cartilage;

Destruction of the alae nasi.

Coexistence of other lupic lesions of nose, cheek and face in general.

Coexistence of other lupic lesions of the mucosa.

Narinal atresia in tunnel form.

2. We can almost, with certainty, include as lupus the cases where one of the following signs is shown:

Long duration of the affection.

Frequent recrudescences in spite of methodical treatment.

Lymphangitis of the lobule.

The presence of vermicular cicatrices on the lobule or at the narinal orifices.

The presence of "eczema" of the nares, which, in several cases, seems to me to have been nothing else than vestibular lupus.

These signs acquire the value of certainty if they coexist with one of the symptoms above mentioned.

The absolute indolence of lupus contrasts with the pain, often sharp, of miliary tubercular ulcerations.

I may add that the rarity or absence of bacilli upon histologic examination is also in favor of lupus.

Let us now examine, in the light of these facts, the cases enumerated in my table.

If we throw out nine case reports which are really too brief to be utilized (Wroblewski I and II, Chiari III, Capart, Silberstein I and II, Herzog V, VI, VII) we see that the cases can be analyzed as follows:

Tumors.	44
Ulcerations	20
Infiltrations	21
Mixed lesions	2

The tumors comprise 29 certain lupus tumors and 1 probably lupus. Only 14 of these cases (which includes Schaeffer's 6) are noted as the only pituitary lesion. I do not hesitate to consider them as lupic tumors; in fact, nothing distinguishes them from other tumors accompanying pituitary lesions; like them, their structure never or rarely revealed bacilli, and like

them, were in patients clear of pulmonary lesions; in a word, nothing authorizes their separation.

Of the twenty ulcerations, eighteen appeared as the ultimate manifestation in pulmonary tuberculosis. Their aspect did not differ at all from granular lesions met with upon any other mucosa (ulcer with scooped out borders, very painful, miliary granulations in a collarette about the periphery) and bacteriologic examination showed the presence of numerous tubercle bacilli. We may conclude that they were terminal granular ulcerations in cases of pulmonary tuberculosis. Two other cases, however (Baurowicz, Michelson II) are manifestly cases of lupus.

In the twenty-one observations with infiltration of the mucosa, only four (Cases III and IV of Hahn, VI of Koschier, and IV of Herzog) are probably lupus; all the others, seventeen, are absolutely lupus. I consider as mixed lesions, granular and lupic, the lesions described in case XVI of Koschier, I of Bar, and the case of Tournwaldt.

The muriform hyperplasia and tumor of the inferior turbinate in Tournwaldt's case, the perforation of the septum in Koschier's case, the smooth pseudo-polyp masses in Bar's case are lupus manifestations as all the other observations testify. They contrast singularly in their slow progress, indolence and absence of ulceration, with the pain of other lesions, the granular ones of the pharynx, larynx and floor of the nasal fossa.

Lupus is slow in evolution, insidious and goes unnoticed until it becomes obstructive. One treats an eruption or a granular ulceration in the pharynx and larynx; in examining the nasal fossae lesions are found which one believes to be contemporaneous and of the same character, but they are of an earlier date and a different sort.*

We see then, to sum up, that there exist only two varieties of nasal tuberculosis: Miliary tuberculosis with frank and unequivocal characteristics, and lupus which comprises: tubercular tumors and the majority of tubercular vegetations. I say the majority, for certain infective granular ulcerations may bud and give rise to vegetations as in the case reported by Band and Tixier, but the granular type of ulcer remains easily recognizable.

The facts seem to me clearly demonstrated: Under the

*I could cite other examples of manifestations described as tuberculosis which are really lupus.

names of tuberculous tumor, vegetating tuberculosis and lupus, writers have described a sole and single thing. This identical thing should be called lupus, for it is characterized objectively and functionally by the same symptoms which, as I have shown already, characterize pituitary manifestations of those having skin lupus.

Kayser (*Monats. f. Ohrenheilkunde*, April, 1901), cites a case of primary nasal tuberculosis in which he described, besides perforation of the cartilaginous septum, the mammillated infiltration characteristic of lupus.

Symond describes under the name of tuberculous ulceration of the septum (London lar. Society, 1897, in *Rev. hebdomadaire*, 1897), a plain case of lupus.

Williams (London lar. Society, April, 1897, in *Rev. hebdomadaire*, 1897, 2, p. 899), describes as common tuberculosis a tumor coinciding with lupic pituitary manifestations.

This study is solely clinical. I will say at once that pathologic anatomy no longer permits a differentiation of lupus from tuberculosis of the nasal mucosa. M. Schmidt (*Die Krankheiten der Oberen Luftwege*, 3rd edit., 1903), after Massei, affirms that tuberculous tumors cannot be differentiated macroscopically or microscopically from lupic new formations.

IV.—ETIOLOGY.

Thus defined, pituitary tuberculosis ceases to be the clinical rarity taught in the classics. If cases of pituitary tuberculosis are relatively rare the number of lupus cases is, on the other hand, relatively considerable. One has only to read, to get an idea, the reports of various scientific bodies and especially the London Laryngological Society. I am even convinced that it is greater still than indicated by the number of lupus cases published. For one thing, lupus is painless, only slightly attracts the patient's attention and may heal of itself; some do not even ask medical attention. For another, very scattered lesions pass unperceived at the beginning.

My statistics confirm the classic figures relative to sex and age of predilection. There is a marked predilection for the female sex, which covers three-fourths of the cases in my list. The ratio is larger still in my personal cases, as I have had eighteen women to two men (90 per cent).

As to age, lupus attacks especially in middle life. By referring to my cases and tables, it will be found that the maximum of frequency was between 15 and 25 years, 44 per cent. There was equal frequency between 5 and 15 and between 25 and 50. Beyond 50 it is very rare. I have had one case at 63 and another at 71.

It always develops upon scrofulous soil. It often succeeds a strumous pseudo-atrophic coryza (Moure).

I believe, moreover, and I have tried to prove that pseudo-atrophic coryza, like atrophic rhinitis (with or without ozena) are larval forms of tuberculosis.

V.—PATHOLOGY.

To the two varieties, granular and lupic, belong two different pathologies.

Lupus is a local tuberculosis, primary, independent of pulmonary lesions. Miliary tuberculosis, on the contrary, is secondary to pulmonary lesions. In lupus the infection is direct; it is brought about by inhalation or direct carriage by soiled fingers. In miliary tuberculosis, on the contrary, the infection is carried from the lungs to the nasal fossae by the blood.

1. *Pituitary lupus is primary.*—All writers upon this subject are not agreed as to this.

Herzog, for example, in eighty cases of nasal tuberculosis only found twenty (one-fourth) where there was not pulmonary tuberculosis at the same time. All his cases of secondary tuberculosis belonged to the ulcerous form. My statistics, made up in part from the same sources as his, show differently.

Francis Steward¹⁸ collated 100 cases of nasal tuberculosis, of which six were personal. He found that the pituitary lesions were primary in fifty-eight, secondary in thirty-seven, and doubtful in five. But he admits that the tuberculous tumor (which he distinguishes from lupus) is most often primary (33 out of 40).

Pistre (tuberculous pseudo-polyps) considers tuberculous tumors to be "most often secondary." This assertion is the more surprising from that author, since in his thirteen cases eight were manifestly primary tumors, three made no mention

of pulmonary conditions and only two show the coexistence of tuberculous tumor and pulmonary lesion.

Pasch,³⁰ while recognizing that the tuberculoma (which he also distinguishes from lupus) is most often primary, has also seen it coincide with tuberculous infiltration of the lungs.

All my personal cases were primary lupus of the pituitary membrane. In no case (my personal statistics include twenty-two cases—sixteen new cases, four published in this journal in January, 1906, and two in November, 1906), did I find a pulmonary lesion.

In the cases collated from the literature, only nine coincided with tuberculous lung lesions (case of Tournwaldt, VI of Hahn, IV of Michelson, IV, VII, XI, XVI of Koschier, I of Hinsberg, and I of Bar).

Now the coincidence of lung lesion and pituitary lupus does not prove that the pulmonary trouble caused it. In none of these cases is the chronology recorded, and nothing permits the affirmation that the lung lesion preceded the nasal lesion. On the contrary, important reasons could be advanced in favor of a pituitary beginning.

We know that lupus makes extremely slow progress (see further on) which is figured by years.

We know, further, that its march is extremely insidious, and consequently that it may have long existed, when it comes the first time for examination. Now all these nine cases report, with slight pulmonary lesions, advanced lesions in the pituitary.

The patient of Tournwaldt presented pulmonary induration, but his nasal fossae were the seat of a muco-purulent discharge since infancy, and the lupus was quite extensive.

The patient in Case VI of Hahn had signs of softening and pulmonary shrinking, but the septal perforation testifies to the duration of the pituitary lesion. The patient in case VI of mine reported January, 1906, had only in 1902 a slight mammillation, the size of a pea in the inferior meatus which would have passed unnoticed if my attention had not been called to it; five and a half years later she had lesions of the cartilaginous septum and anterior thirds of both inferior turbinates, without perforation of the cartilage. No pulmonary lesion.

In Case IV of Michelson it is expressly noted that the patient dated his nasal obstruction several years back.

The same in Cases VII and XVI.

On the one hand slight lung lesions; on the other, advanced pituitary lesions. Logic holds that the latter began first. Moreover, these cases constitute an insignificant minority.

In fact, if we deduct twenty-one cases from the table where the pulmonary condition was not shown, we find thirty-seven cases of primary lupus to which must be added my sixteen personal cases and the six others published in January and December, 1906, so that there are fifty-nine cases of primary lupus against nine of lupus with pulmonary lesions. The primary lupus cases make, then, 90.5 per cent of the whole.

Another consideration, indirect but important, confirms me in my opinion. As Francis Steward¹⁸ has remarked, if we add together the tuberculous lung cases of Willigh¹⁹ and Weichselbaum,²⁰ we get a total of three cases of pituitary tuberculosis in 640 lung cases, that is, 0.46 per cent.

This shows that in coincidence of pulmonary and pituitary tuberculosis it is the nasal tuberculosis which came first.

It is not rash to think that the twenty-one cases in my table who had no mention of the pulmonary condition were primary pituitary lesions.

2. *Granular tuberculosis is, on the contrary, secondary to a pulmonary lesion.*—This appears from an examination of the cases reported by my colleagues in this essay. All the granular ulcerations therein recorded appear to me to be a terminal lesion in the course of pulmonary phthisis. There is but one case of granular ulceration of the pituitary membrane without a pulmonary lesion; this is the one described by Melzi²¹ in the *Archives Internationales de Laryngologie*, a patient who came to him for a very pronounced nasal obstruction. The author made out a common hypertrophic rhinitis and did a turbinotomy. To his great astonishment he found on the meatal aspect of the inferior turbinate an ulceration comparable to ulcerations on the tongue. The borders were undermined. On the periphery were several characteristic gray granulations. The patient was young and clinically free from pulmonary tuberculosis. The ulceration was found histologically to be tuberculous but no Koch's bacilli were found.

This case is unique to my knowledge. It does not contradict the rule that a granular ulceration is most often a terminal phenomenon in the evolution of a pulmonary phthisis.

It is a blood infection. There is a bacillary discharge into

the circulatory system from the pulmonary parenchyma which becomes colonized in the nasal fossa. Usually there is a massive bacillary discharge. One finds in these ulcerations numerous Koch's bacilli, contrariwise to what is found in lupus lesions where the bacilli are rare or undiscoverable. To sum up; there exist two modes of pituitary infection: the blood route producing miliary tuberculosis; the air route causing the appearance of lupus.

Escat supposed in the latter event, that the pituitary mucus had the power in some way of attenuating Koch's bacilli, modifying their virulence. The nasal fossae would thus be a sort of physiologic laboratory for the attenuation of bacilli.

VI.—SYMPTOMATOLOGY.

I have had especially in view in this study primary tuberculosis, lupus of the pituitary. I ought to say, however, a few words about granular tuberculosis.

A.—Miliary Granular Tuberculosis.

This presents in two forms: gray granulation, and ulceration. Gray granulation is extremely rare in the pituitary mucosa. It appears in the form of an eruption of small granulations, the size of a millet seed, gray, separated from each other by areas of healthy mucosa. They are usually seated upon the lower turbinate. If a swab culture be colored and examined a considerable quantity of tubercle bacilli will be seen.

I have found but two cases: One in the thesis of Olympitis which belonged to Millard, and another in Boutard's thesis which belonged to Hajek.

In the latter case the gray, translucent granulations were seated upon the inferior turbinate; a slide examination showed numerous tubercle bacilli. They went on very rapidly to ulceration. This rapid transformation is the rule, and doubtless explains the infrequency of a pituitary miliary eruption.

Granular ulceration shows nothing peculiar. It has the usual aspect of granular ulcerations as seen upon the tongue (undermined edges, collarette of miliary tubercles). It has a marked predilection for the anterior portion of the nasal

fossae, the anterior part of the septum and the neighboring part of the upper lip. It is an ulceration almost as much narinal as nasal, and this perhaps explains the sharp pain accompanying this lesion as opposed to the remarkable indolence of lupus.

Granular ulceration of the pituitary mucosa has, then, a real existence, and, by its location, its character, the pain which it produces, the numerous bacilli in the scrapings, the co-existence of pulmonary phthisis, clearly distinguishes itself from lupic ulceration. It may, especially if infected, become vegetant, but it rarely goes on to perforation of the cartilage.

B.—Lupus.

I have given, at the beginning of this paper, the clinical picture of lupus at its height. There remains something to be said of its mode of onset, its functional symptoms, its course, and the condition of the facial lymphatic system.

1. *Mode of onset.* The pathognomonic signs of pituitary lupus.—I have already indicated the modes of onset in my work of January, 1906. Repeating what I have said there are:

A form of lupus with a lacrymal beginning. A form with a lobular lymphatic beginning. An impetiginous vestibular form.

I refer to this work for the details of these modes of commencement. I will merely say that these modes appear to me to have much practical importance. They deal with the external phenomena immediately visible to every practitioner, not requiring the special manipulations of the rhinologist. They are the revelatory symptoms of pituitary lupus. If the practitioner knows that lacrymation, or a rebellious impetigo of the vestibule, or a torpid and recurring lymphangitis of the lobule may be the index of a baffling nasal tuberculosis, he will have him examined by a rhinologist without delay for the maladies coming under this category, and thus have his patient avoid a long lack of treatment which might result in a lacrymal fistula, destruction of the lobule or the subseptum, ending in the horrible nose destruction called the "death's head" or "parrot beak."

2. *Functional symptoms.*—These are almost nil unless the lesion becomes obstructive. It is even probable that it may remain latent for a long time.

Then there appears a dryness in the nose, or rather a drying of the secretions in the form of crusts which are sometimes sanguinolent. No pain, neither spontaneous nor upon pressure. Then the lesion becomes vegetating, or, if the crusts are abundant, nasal obstruction begins. Little or no reaction upon the general health. Sometimes the patients look really florid.

3. *State of the facial lymphatic system.*—I have already dwelt enough upon the local symptoms. I wish to say only a few words as to the lymphatics of the face.

It is known that the pituitary lymphatics (Andre, "Les lymphatiques des fosses nasales," Th., Paris, 1905) follow two currents of opposite direction.

The lymphatics of the mid-posterior portion are directed backward and terminate by a superior trunk at the retropharyngeal ganglia and upper ganglia of the jugular chain; by a middle trunk at the subdigastric and jugular ganglia (the former of which also receives the lymphatics of the anterior route); and by an inferior trunk at the ganglia of the jugular chain near the bifurcation of the carotid.

The lymphatics of the anterior third of the pituitary membrane participate in the distribution of the nasal integument. They therefore present three collecting groups; a middle group which ends at the submaxillary ganglia, following the path of the facial vessels; a superior group which ends at the preauricular and parotid ganglia; and an inferior group to the sub-mental ganglia. Along the facial chain are found a number of very small ganglia, seen in twenty out of thirty-two cases by Poincetreau, who pointed them out; the inferior, situated upon the horizontal branch of the inferior maxilla, in front of the masseter; the middle on the external aspect of the buccinator; and the superior, comprising a naso-genial and a suborbital ganglion.

Theoretically, gland infection ought then to be systematic as follows: To a lesion in the posterior two-thirds of the mucosa would correspond a retropharyngeal or retrodigastric adenitis; to a lesion of the anterior part would correspond an inflammation of one or more face ganglia.

Retropharyngeal adenitis has not been reported and I have not seen it; but a slight adenitis could go unnoticed by reason of the difficulty of examining this region. Moreover, at the beginning the lesions are confined to the anterior part of the

pituitary membrane. All the ganglion territories of the face have, on the contrary, been involved.

Personally I have noted: Submaxillary adenitis (Cases IV, V and VI); subdigastric adenitis (Case VII); cervical and submental adenitis (Case VIII); submaxillary and carotid (Case X); submaxillary, submental and premaseteric (Case XV); premaseteric (Case XVI); submaxillary and carotid (Case I, *Memoirs* of September, 1906).

I have noted in the histories which I have looked through: Suppurative preauricular adenopathy, with subangulo-maxillary adenitis (Case XII of Pistre); submaxillary adenitis (Herzog, III and IV; Chiari, II; Silberstein, I and II); submaxillary and cervical adenitis (Pistre, XI; Hahn, II and VI); adenitis of the cervical glands (Hinsberg, IV). I recall also the two cases of Gelle and Poulard (Th. of Poulard²³).

I have seen along with a unilateral nasal lesion bilateral adenitis, a fact which anatomy well explains (crossed distribution of lymphatics).

Apparently not much attention has been given to the condition of the lymphatic system in nasal tuberculosis. In eighty cases which I have collated I have found adenopathies in only ten, usually submaxillary. Andre in the clinical part of his thesis notes only the case of Poulard, previously mentioned. Gelle in his work²⁸ on adenopathies of nasal origin, remarks that writers are mute upon this question not only as to tuberculosis but also as to lupus, most authors making this distinction. However, it appears to me to be frequent, for I have found it in ten of twenty-two cases, or 45 per cent. I believe it is even more common than the figures indicate, for at the beginning, I did not systematically explore the regions where I might find adenitis.

This examination of the ganglia has some diagnostic import, for, if along with adenitis there is no skin lesion, one will be led to examine the nasal fossae and perhaps find lupus lesions at their beginning.

Above all, as we shall see, it has pathologic importance. As the pituitary lymphatics follow the course of the tegumentary lymphatics, one may ask if the adenopathies which I have found with relative frequency, as remarked, are not due to skin lesions which sometimes coexist with pituitary lesions at the time when the patient comes for examination.

There is nothing in this. For if in a small number of cases

the adenopathy does coincide with the lesions of the nasal integument (as in Cases V and X and Case IV of January, 1906), nothing goes to show that the adenopathy did not precede the cutaneous lesion; and if, in others, an interpretation of the adenitis is complex because of concomitant lesions (Case II, September, 1906), in the very large majority of cases (seven out of ten) the adenitis appears in the course of a primary pituitary lupus. Facial adenopathies, therefore, are evidence of the pituitary lesion and should cause it to be sought for.

4. *Progress, duration, termination, prognosis.*—The progress is extremely slow, torpid and insidious. It may exist six years (Case VII), eight years (XII), or ten years (V) without revealing itself except by insignificant symptoms, or simply by lacrymation. This slowness of evolution may be still greater, as witnesses Case X of Koschier. This was a woman of 71, who, together with a septal perforation, had some other small pituitary lesions. Twenty years previously she had been treated for a septal neoplasm; for twenty years then the lupus had remained limited to the anterior part of the nasal fossae, without having progressed.

Hence the duration of pituitary lupus is to be measured by years. Sometimes, however, probably as a result of secondary infections, it takes a more rapid course. I allude to those cases of lupus vorax, which in a few weeks destroy the cartilaginous septum, the subseptum, and the lobule of the nose; the diagnosis from syphilis may give difficulty in such cases. An important feature in the course of lupus consists in frequent recurrence as all cases show.

Lupus has also some tendency to spontaneous cure by reason of fibrous tissue production in the lesion which here, as everywhere else, tends to smother the lesions which it surrounds. It is true that, while it often heals in one place it breaks out afresh in another, in the cavum or in the larynx as we shall see. However, the formation of this fibrous tissue, the presence of which Garel and Collet²⁰ used in distinguishing histologically lupus from active pituitary tuberculosis, has the result of bringing on an atrophy of the inferior turbinates; and in an advanced lupus (especially if the lesions predominate in the inferior turbinates) it is common to see the nasal fossae considerably enlarged by a sort of lupus atrophic rhinitis, a novel proof of the role of tubercular toxins in produc-

ing turbinal atrophy. But if lupus does heal sometimes spontaneously it sometimes resists the best and most persistent treatment.

In a certain number of cases the lupus patient does not get well and dies of pulmonary tuberculosis. I have seen this eventuality once. Thus, while the patient with pulmonary tuberculosis never, so to speak, becomes lupic, the reverse is far from rare (Lenglet).

All these conditions show us that nothing is more difficult than to predict the future of a pituitary lupus, and fully justifies these words of Massei: Fears and hopes make up the prognosis; hopes in view of the benignity and slowness of the malady, permitting radical intervention; fears because of the complications which may involve other organs. Sometimes, after several years of reasonable hope, there come localizations in the lung, the larynx or the meninges, with a rapid course, carrying off the patient who was considered cured.

VII.—COMPLICATIONS.

If we can compare the nasal fossae to a laboratory where Koch's bacilli are attenuated, we must also recognize that this same laboratory, after attenuating them, spreads them in every direction. Situated right at the center of the face, at the beginning or end of many organs, it is not astonishing for the nasal fossae to scatter bacilli into these different organs.

Forward we see the pituitary membrane continuous by insensible transition with the skin of the face. In the inferior meatus it is continuous with the lacrymo-nasal canal; in the middle and superior meatus with the various sinuses of the face.

Behind, it continues, the cavum being intermediary, into the mucosa of the Eustachian tube and the tympanic cavity, and of the pharynx, esophagus and larynx.

Having so many paths which the bacilli may follow up, we have so many varieties of complications to study.

A.—Cutaneous Complications. Relations of Pituitary and Facial Lupus.

At first no account was taken of pituitary lesions in producing cutaneous lupus. The pituitary lesions were

considered to be due to the spread of skin lesions to the nasal mucosa. Then it was recognized that lupus of the nasal mucosa could appear without any preliminary skin lesion. Primary lupus had acquired citizen's rights (*Th. of Raulin*, 1889). In spite of this, probably on account of the dominance of old notions, nobody saw that lupus of the mucosa could spread to the skin. Raulin himself believes that facial lupus is usually a primary skin infection, while, exceptionally, it is caused by a pituitary lesion.

The more and more frequently established coincidence of pituitary lesions with facial lupus forced a study of their relations, and it was asked if, instead of being an epiphenomenon in the course of facial lupus, the pituitary lesion was not the source of the cutaneous infection, and consequently the essential thing.

Rhinologists and dermatologists defend both opinions. The classic authorities believe rather in a primary cutaneous infection.

Lupus of the nasal fossae, says Lermoyez (*Therapeutique des maladies des fosses nasales*) may be: (a) primary when it begins in the pituitary membrane; (b) secondary, when, which is ordinarily the case, it first attacks the skin of the face and subsequently enters the nasal fossae.

Garel (*Diagnostic et traitement des maladies du nez*, 2nd edit.) after first believing in the rarity of primary pituitary lupus, now considers it relatively common, but, besides lupus of the nasal integument by spread from pituitary lupus, he believes in a primary lupus of the nasal integument whose nodules are much more destructive than the nodules of the former variety.

Menier²⁴ in his excellent manual thinks that most often when cutaneous and mucous lesions coincide, the former appeared first. On the other hand, Chiari²⁵ often remarks in the anamnesis of numerous cases of facial lupus, that these individuals, long before the appearance of the lupus, had a sore at the nasal entrance.

Bresgen²⁶ believes, as Neisser did before him, that facial lupus, as a general rule, is only an extension of a mucosal lupus.

Escat (cited above) thinks that cutaneous lupus is always consecutive to a pituitary lesion.

Molinie²⁶ believes that primary pituitary lupus is relatively frequent.

Audry²⁷ thinks that "skin lupus is usually if not always secondary to initial tubercular foci situated upon neighboring mucosa or deep tissues."

Sticker²⁸ professes the same opinion. Meneau and Freche²⁸ found in twenty-five cases of facial lupus, twenty-one where the nasal mucosa was the prime cause. There had been no rhinologic examination, the authors basing their opinion on the patient's nasal history (hypersecretion, crusts). They are convinced that "the frequency of lupus with a pituitary beginning, would be still greater, contrary to general opinion, if the nose were examined systematically and if nasal lupus could be detected at the outset." At the same time they do not consider lupus at the center of the cheek as being consecutive to pituitary lupus.

Lenglet (*Pratique dermatologique*) admits two methods of producing cutaneous lesions: (1) Cutaneous inoculation (kissing, scratching, former dermatoses). (2) Propagation from a lesion in the nasal mucosa. He seems to believe, with Raulin, that primary cutaneous lupus is the most frequent mode. Like Meneau and Freche he does not link lupus of the face, other than the nose, with pituitary lupus.

Professor Gaucher in his *Traite de dermatologie* is eclectic; various modes of pathogenesis of facial lupus are possible.

As for myself, I am firmly convinced of the pituitary origin of lupus of the nose and face.

This opinion on the one hand is based upon case reports which I consider unassailable; and on the other hand is the only way of explaining all the topographic modalities of facial lupus.

If certain dermatologists do not admit the pituitary portal of entry, it is because they do not understand very well the bond of union between the nasal lesion and the cutaneous nodule. "If it is right to regard lupus of the alae nasi as the result of a mucosal lupus, it is less evident that there is always a relation between lupus in other facial regions and lesions of the neighboring mucosa." (Lenglet, *loc. cit.*)

This bond of union is the lymphatic system.

We have now to study the case reports upon which I base my opinion, and the role of the lymphatics in the appearance of facial lupus.

1. *Facts which demonstrate the pituitary origin of nasal and facial lupus.*—My twenty-two observations of pituitary lupus may be divided into three classes: (a) Pituitary lupus without cutaneous lesion; (b) cutaneous lupus coinciding with pituitary lupus; (c) cutaneous lupus coinciding with a non-specific pituitary lesion.

Of the first class I have nothing to say, except that it was not necessary to wait for an outbreak of cutaneous nodules to make the diagnosis of pituitary lupus.

Of the second class I will say that in all cases where it existed, the pituitary lupus plainly preceded by several months, sometimes by several years, the cutaneous lupus outbreak.

The third class is represented by one case of lupus of the cheek coinciding with a rhinitis, atrophic in type without ozena, and which I reported in the *Annales* of January, 1906.

Here my view seems at fault. There is, then, at least one case of facial lupus without any pituitary lesion. Now it is not proven that an affection so little understood as atrophic rhinitis is not a manifestation of tuberculosis. I gave to the French Laryngological Society of 1907⁴⁰ the clinical reasons favoring this hypothesis. Lombard, to whom I imparted my ideas as to the possible tuberculous nature of atrophic rhinitis, has obtained a general and local reaction by an injection of tuberculin in a patient having ozena. It is then not impossible that atrophic rhinitis may be a sclerotic tuberculous manifestation of the same sort as certain renal scleroses (Barnard and Salomon) and possibly hepatic scleroses. But, even if this opinion is not irrefutably demonstrated, the case of lupus of the cheek of which I have just spoken must be regarded as of pituitary origin.

We shall see further on that pituitary lupus spreads to the face by the lymphatic route. Now, it is perhaps not necessary for Koch's bacilli to produce a lesion at the point of inoculation in order to go thence to the lymph relays of the face. This explanation is not at all contrary to the general laws of pathology. It is positively known that Koch's bacillus penetrates the intestinal wall without making any lesion in it, and infects the mesenteric lymphatics, possibly also thus going from place to place in the lung. It is known that this same Koch's bacillus penetrates the tonsils in the same way, infects the tributary glands (Dieulafoy,⁴¹ Babes⁴²) and eventually the lung.

It follows from this that facial lupus would always be of pituitary origin, whether or not there be any lesion at the port of entry.

2. *Role of the lymphatic system in the appearance of facial lupus.*—It is curious to note that the places of election of facial lupus are just those points where there are ganglia, or lymphatic relays: Cheek bone (suborbital ganglion of Poincetreau); cheek (genial ganglion); horizontal branch of the inferior maxilla (pre-masseteric ganglion), these are for the lymph chain of the facial vessels. Ascending branch of the inferior maxilla (preauricular ganglion), submental region (submental ganglion).

A priori we would have a right to infer that the skin is infected through these ganglia or efferent vessels acting as intermediaries. Observation shows that just these ganglia are very frequently attacked in the course of pituitary lupus. I have long insisted upon this fact. We may conclude, then, that these ganglia play an essential role in the appearance of cutaneous lupus.

Arriving at the ganglion the bacillus may infect the skin and cause lupus in two ways:

(a) It may produce a suppurative adenitis, the fistula from which is the focus of the lupus (Cases VIII and XIII, lupus around a submaxillary adenitic fistula). The role of the lymphatic system is especially clear in the latter case. In fact, although the patient declared that the lupus nodules on the nasal lobule had been contemporaneous with the nasal obstruction and nasal crusts, the extent of the pituitary lesions proved that they came well before the cutaneous lesions. Now, considerably before the appearance of lupus in the lobule, the whole lymphatic chain of the facial vessels had been infected. Three years before my examination there was a suppurative right submaxillary adenitis treated surgically, and a suppurative submental adenitis. Ten months before, right suppurative pre-masseteric adenitis. Three or four days before my examination, appearance of a tuberculous gummi in the skin, the size of a small pea, at the center of the cheek.

(b) The ganglion does not suppurate but the bacillus infects the afferent cutaneous lymphatics. I have been able in one case to find at the center of a lupus in the masseteric region (progressing to a cure, by the way) a small mobile ganglion, palpable proof of the mechanism of infection.

It is very evident that if instead of the ganglia described by Poincetreau, there exists only a lymphatic plexus, the latter would play the same role as a ganglion. If it were necessary to give other proofs of my ideas I could relate several other cases which I have had the good fortune to observe and which have the weight of laboratory tests. I have already related one of these cases in my note of January, 1906 (Case IV).

This was a young girl of 13, who, some time after a curettage of a lupus in the vestibule, noticed a lupus nodule appear on the left cheek bone, while at the same time, upon the horizontal branch of the inferior maxilla, same side, exactly at the spot occupied by the premaseteric ganglion, appeared a tuberculous subcutaneous ganglion, still mobile but rapidly adhering to the skin.

The second case, in every point comparable to the preceding, is that described as Case VI in this report. Five days after a nasal curettage for a vegetating lupus there appeared redness of the cheek with characteristic lupus nodules, which were treated as such at the Hospital St. Louis, where I sent the patient. I have been able to see counterparts of these cases.

If a curettage of the mucosa can inoculate the skin of the face, inversely an amelioration of the nasal lesion can bring on a spontaneous disappearance of the skin lesions. This is what happened in the case above cited where the face lesions (lupus nodule and gumma) diminished gradually and disappeared without any special treatment other than that directed to the nasal mucosa.

Bresgen³⁰ has reported analogous cases of disappearance of face nodules after endonasal treatment. I have noted a complete concordance between the pituitary condition and that of lesions of the nasal integument, the latter growing better and even disappearing with pituitary improvement, reappearing when on account of insufficient care the pituitary lesions resumed their progress (Case III, *Annales*, January, 1906). Hence we can say that the state of the skin of the nose and face reflects the state of the nasal mucosa.

I have especially in view here lupus of the face, but it is very evident that lupus of the lobule is produced through the lymphatic openings in the skin.

I may add, in closing, that lupus may be inoculated into the skin without the medium of lymphatics. It sometimes fol-

lows the lacrymal canal and comes out upon the face through a fistula of the sac (Case II, *Annales*, September, 1906. See also cases of Hinsberg). Nevertheless this mode of propagation is more rare than the other.

It is not my intention to deny the possibility of direct infection of the skin. Clinically a previous skin lesion (eczema usually, but we must mistrust those eczemas which are sometimes tubercular manifestations) may favor contagion carried by kissing or rubbing.

Experimentally the bacilli can be made to enter the intact skin by friction (Babes,⁴² Courmont and Lesieur⁴³). But, for one thing, these experimental conditions are never realized clinically. For another, direct infection of the skin cannot explain the clinical peculiarities of facial lupus, especially its topography, which the lymphatic pathogenesis I advance explains so clearly.

B.—Lacrymal Complications.

Infection of the lacrymal passages in the course of pituitary lupus presents a quadruple character: frequent, early, caused by a simple or by a specific infection, tuberculous like the lesion which gave it origin.

It is frequent.—In twenty-two cases of pituitary lupus I have seen it eleven times. If I add two other cases of lupus which I have seen with lacrymal lesions and which were sent to me by Dr. Morax, we have a total of thirteen cases of lacrymal infection out of twenty-four pituitary lupus cases, or more than 50 per cent. I am not a little surprised to see this cause of lacrymation so infrequently mentioned in the French works upon lacrymal infections.

Raulin declares epiphora to be possible theoretically but he has not found it mentioned in the sixteen cases which he has collated.

Herzog notes several cases in his memoir.

Gelle²² in his report to the French society noticed nasal tuberculosis but twice in 100 cases of tearing examined by him in Prof. Lapersonne's clinic. He cites statistics of thirty cases of tearing collated by Faravelli and Kruch without a single case of tuberculosis.

It is early.—In my eleven cases it appeared twice even before any nasal trouble had attracted the patient's attention; three times simultaneously with the nasal complaint, three

times six months afterward, and once eighteen months afterward. In two cases the time of appearance was not indicated. Infection is therefore clearly contemporaneous with the beginning of the nasal lesion, and is indeed, as I say, a revelation sign. It is possible that the slighness of the lesion has caused it to go unperceived in a number of cases reported.

It may or may not be specific.—If it is a simple infection, inferior meatoscopy will show no lesion in the meatus proper. The lesion stops at the meatal floor. Or, it produces a synechia of the inferior turbinal border with the floor, favoring a relative stagnation of the tears and an ascending infection of the lacrymal passages.

On the contrary, if it is a tubercular infection of the lacrymal passages, meatoscopy often reveals characteristic lesions in the inferior meatus. Thus we may find a small lupus tumor in the meatus, seeming to come from the infundibulum (Cases I and II, *Annales*, September, 1906). Sometimes, too, the meatal surface of the inferior turbinate is involved, and fuses with the sinus wall, partly obstructing the inferior meatus. But the absence of a meatal lesion on meatoscopy does not absolutely prove that the lacrymal infection is of the simple sort. I have seen a case of lacrymation without a meatal lesion, which I first considered to be secondary and without significance, give rise later to fistula of the lacrymal sac and a lupus of the overlying skin.

It is therefore difficult to say in a case of tearing whether the lacrymal infection is simple or tubercular, and whether or not the patient is menaced by lupus of the sac integument.

C.—Sinus Complications.

Infection of the sinuses is possible in theory, and, as in the lacrymal passages, we can conceive of a simple infection of the frontal or maxillary sinus, manifesting itself by a catarrhal sinusitis, or a specific tubercular infection of the sinus mucosa.

I have never found signs of catarrh about the middle meatus. On the other hand, endoscopy would evidently not be informing as to the state of the sinus mucosa, for the infiltration of the mucosa would not be sufficient to intercept the light rays. I have used transillumination three times for the maxillary and frontal sinuses without getting indications of any lesion.

In fact, tuberculous sinusitis is not thus produced; it is always consecutive to a tuberculous osteitis adjacent.

However, Gleitsmann,³³ who has collected twenty scattering cases of sinusitis from the literature, reports (though without giving details) two cases of maxillary sinusitis resulting from pituitary lupus. Gradenigo at autopsy of a tuberculous lupus patient found the mucosa of the frontal and sphenoidal sinuses infiltrated and swollen (*Ann. des Maladies d'Oreille*, 1899, p. 93).

To sum up, the complication appears to be rare and will not hinder you.

D.—Aero-digestive Passages.

Localization in the cavum is an intermediate step by which lupus extends to the Eustachian tubes, the middle ear, the pharynx, velum and larynx. I will not expand upon lupus in these regions. That would be going beyond the scope of my subject.

I will only remark that these localizations are absolutely painless, having no symptoms or almost none. Hence it is necessary to examine these regions systematically in order to determine any extension thereto.

E.—Aural Complications.

The action of lupus upon the mucosa of the ear is exactly comparable to that upon the lacrymal passages. It may act in two ways:

(a) It occasions a simple infection which shows as a catarrh of the middle ear, exactly analogous to the lacrymation previously mentioned.

(b) Or it invades the tube and middle ear, produces lupus of the middle ear, dislocates the chain of ossicles, perforates the tympanic membrane and shows itself to the patient by a slight discharge from the canal.

The ear is frequently involved, for Felix³⁴ in twenty lupus cases seen at the dermato-syphilitic clinic of Bucharest found such lesions in every one; seventeen catarrhal and three suppurative.

The lesion must be sought out, for the impairment of hearing is often not enough to attract the patient's attention.

I have frequently found middle ear catarrh, and once (Case

XII) suppuration of the middle ear with total destruction of the drum except Shrapnell's membrane.

The prognosis is grave. On the one hand, treatment of the catarrh is often made difficult by the lesions in the nose and pharynx, partly because of the difficulty of introducing sounds and partly because of the constant reinfection of the tympanum. On the other hand, lupus of the tympanum may spread. Thus Gradenigo³⁵ has reported a case where the lupus had not only destroyed the ossicles and invaded the mastoid mucosa but had penetrated the labyrinth by breaking through the round window, occupying all the tympanic area. It had also invaded the oval window, and a little of the external semicircular canal. It was, as may be seen, a genuine lupus vorax of the ear. The type of lupus was very severe, having invaded the nasal fossae, the cavum with adhesion of the velum, the tongue, pharynx, larynx, and skin of the face, ear, body and limbs.

F.—Intercurrent Complications.

The most common is erysipelas. It occurs rather often in nasal lupus. Not uncommonly the patient may have in the long evolution of his case, several recurring attacks of erysipelas.

In a number of cases an amelioration of the lesions follows.

VIII.—DIAGNOSIS.

The diagnosis of lupus will be kept specially in view in this chapter.

Miliary tuberculosis has characters (form of ulceration, borders, vestibular seat, peripheral nodules, lively pain, rapid progress, advanced lung lesions) upon which I have dwelt sufficiently, and which in general are enough to make recognition easy. In what follows I suppose an isolated pituitary lupus, since a concomitant face lesion facilitates the diagnosis.

We may have to make a diagnosis in the presence of the following different conditions: beginning lupus, infiltration, perforation, tumor, vegetant lupus.

A.—Beginning Lupus.

The diagnosis is made by recognizing the elementary lesion. Remember that it is necessary to search systematically when a patient has one of the revelatory signs which I have pointed out, and to search in the places of election, upon the cartilaginous septum, the entire anterior portion of the subseptum and the floor of the meatus.

A very small error is possible; one must not take for a beginning lupus a swelling which is sometimes seen at the entrance of the inferior meatus, vaguely mammillated, due to a thickening of the maxilla. It will suffice to note that the mucosa which covers it is smooth and not roughened as in lupus.

B.—Lupus Infiltration.

This is very special; the mammillated infiltration is generally sufficiently characteristic. Several causes of error, though, are to be noted:

1. *Mammillated appearance of old nasal catarrhs.*—From time to time are seen old nasal catarrhs, with a kind of dermo-papillary transformation, giving a mammillated aspect to the floor and turbinates. But, as in the preceding case, the mucosa preserves its smooth, polished appearance; further, it is not infiltrated and nowhere are there adhering crusts.

2. *Diffuse syphiloma of the nasal fossae.*—This is a variety of tertiary syphilis characterized by an often considerable infiltration of the septum and inferior turbinates, which really could not be confounded with lupus infiltration. The latter is mammillated, quite regularly mammillated; its progress is insidious and slow; it causes no annoyance to the patient except blowing out crusts. The nasal obstruction comes on with extreme slowness, often several years after the patient first noticed the crusts.

The infiltration of a diffuse syphiloma has opposite characters to those just described. It is smooth in its ensemble; there are only simple swellings, widely separated from each other, and a few ulcerations when breaking down begins. It comes on abruptly. In two or three weeks the septum and turbinate are quadrupled in volume, causing considerable nasal insufficiency. There is intense headache. Finally, the bones themselves are involved in the syphilitic process and are tender to pressure.

C.—Lupus Perforation.

Diagnosis may be required either of a lupus perforation in progress or of a healed perforation.

1. *Syphilitic perforation* is not easily confounded with lupus perforation. In lupus the perforation is seated only on the cartilaginous septum; in fact tuberculosis always respects the osseous structures. Of variable dimensions (a pea to a two-franc piece or larger), it is surrounded by a collarette of soft bleeding characteristic fungosities. Finally and above all it is never alone, the neighboring parts of the septum, the turbinates or vestibule, show the characteristic mamillation.

Syphilitic perforation is almost always in the bony septum. At that period rather often there are ulceration which upon being explored reveal denuded bone or sequestra. To this may be added the peculiar odor wholly unknown to lupus, the pain, headache, etc.

Exceptionally syphilis may attack the quadrangular cartilage. Considering the relatively rapid march of the lesion it is more likely to be confounded with lupus vorax. In both cases the perforation is rapidly effected. There may be a collarette of fungosities about the perforation and the lobule of the nose may be red and infiltrated. This is certainly a very embarrassing condition.

The diagnosis of lupus is to be made if there is a zone of mamillation about the perforation, if with the lymphangitis of the lobule there are characteristic nodules, if the gland territories of the nasal mucosa are the seat of adenitis.

But secundo-tertiary eruptions may simulate nodules of the lobule; there may be an adenitis in syphilis if there be a superinfection. So that often recourse must be had to specific treatment in order to settle the question.

When the perforation is cicatrized confusion with Hajek's ulcer is possible.

To tell the truth there is hardly any difficulty unless there be no other pituitary lupus lesions. Even in this event the borders of the ulcer would be a sufficient indication. In simple ulcer they are evenly thinned from the periphery to the center, and thin as paper immediately around the hole. In lupus they have a polymorphic aspect, thin in one place and thick in another, as though punched out.

D.—Lupus Tumor.

It is classical to say that the diagnosis of tubercular tumor is difficult, and that it may be confounded even with malignant tumors, especially sarcoma.

To my mind the difficulty is not so considerable. Distinction should be made according to whether the tumor is or is not accompanied by perforation of the cartilaginous septum.

1. *The tumor is accompanied by perforation of the cartilaginous septum.*—There should be no hesitation, it is lupus. Tumor and perforation were caused by the same thing. Only lupus can give this combination.

2. *The tumor is not accompanied by perforation of the septum.*—I repeat what I have just said; it is exceptional for the tumor to be the only manifestation without any other lupus symptom.

There is a hypertrophic variety of secondary pituitary lesion which has been described as condyloma of the nasal fossae and of which two cases have been reported by Lacoarret²¹ and Labit.²²

These growths do not seem to have a very clear symptomatology. In Lacoarret's case the tumor, which was implanted upon the septum, had dimensions of 1 cm. It was composed of three big mammillae and coincided with mucous plaques in the mouth and balano-preputial groove.

In Labit's case there were no concomitant syphilitic manifestations, but the tumor was hard and of horny consistence. Both tumors healed under specific treatment.

These two cases are rarities; they are the only ones I have found in the French literature. Hence there is no reason to give them an exaggerated clinical importance. Nevertheless they should make us prudent in diagnosis, and in case of doubt administer specific treatment.

All unite in saying that lupic tumor may be confounded with malignant tumor of the septum.

The question does not come up unless the patient is old (epithelioma) or when, with a middle-aged patient, specific treatment does not cause any change, the tumor not being accompanied by any other lupic lesion.

The latter condition is extremely rare. But when it exists it is very certain that, no more than other tumors of the nasal fossa, the lupic tumor has no objective character peculiar to

itself. Then the microscope only, or, if necessary, an inoculation, can solve the question.

IX.—TREATMENT.

I am convinced, from the cases which I have treated, that general treatment is of the highest importance. It will not suffice alone to cure pituitary lupus but it puts the organism into a state where it can fight against the infected remnants which local treatment almost of necessity leaves behind, even after a most painstaking curettage. Cases are reported of curing lupus which had local treatment regarded as insufficient by the physician, but which several years afterward were seen in a state of cure under the sole influence of general treatment. Of course we cannot always count upon such marvelous results. But, I repeat, the first indications in treatment are superalimentation, fresh air and cod liver oil.

This said once for all, let us see what local methods we have at our disposal.

The methods employed can be classed as:

Physical.

Chemical.

Surgical.

Physical methods.—They comprise radiotherapy, hot air, phototherapy and cauterization.

Radium therapy.—This method is still in an embryonic state. It has been used by Botay¹⁵ in one case. The method is too new to be passed upon. Of course it can only be used in the big cities.

Hot air.—Hollander ("Traitement de la tuberculose des muqueuses par les cauterizations a l'air chaud," Berlin Society of Medicine, May 16, 1906, in *Presse Medicale*, 1906, p 369), declares that he has gotten excellent results in this way. Combined with general hygienic and dietetic treatment, it will in the majority of cases, even the most advanced, almost always bring on a cure.

I have used this method only once, that was in Case VI. Treatments were given every eight days for a month, and hot air was applied to the lesions for five minutes. I then applied the canula directly to the lesions and made applications of six to eight minutes, hoping thus to get a cauterizing effect. After a month of this treatment the lesions were not influenced at all and I quit treatment. Other similar trials are of course necessary to judge of the method.

Galvano-cauterization.—This is an excellent method if too much is not expected of it. It is indicated in beginning lupus when the lesions are limited to the septum or the inferior meatus or the end of the turbinate. It is wholly insufficient for vegetant lupus.

Here is my method: Using a galvanic knife I make burns upon the infiltrated region, parallel to each other and covering the entire affected region. Over these cuts and at the same sitting I make another series of rays, parallel to each other, but perpendicular or oblique to the first cuts. This method, which is analogous to that employed by veterinarians for the treatment of joint tuberculosis, has given me better results than cautery points. The burn ought to be deep enough to go through the lesion.

Chemical methods.—Among these some are used directly, without any preliminary treatment of the lesions; others are used only after curettage. Among the first is cauterization with iodid of mercury; among the second is cauterization with lactic acid.

Iodid of mercury.—This proceeding has recently been recommended by Hollander (Berlin Society of Medicine, May 16, 1906).

He uses iodid of mercury in the nascent state. A quarter of an hour before the local treatment he has the patient take a dessert spoonful of 5 per cent iodid of potassium. Then he applies powdered calomel to the lesions, either on a tampon or by insufflation, or an oily emulsion. The iodine eliminated from the mucosa combines with the calomel and gives rise to iodid of mercury, whose action upon the lesions is most energetic. He has used this method not only in pituitary tuberculosis but also in buccal, vesical and laryngeal tuberculosis—the last with fine success. I have tried it in Cases I and VI. without any success. Maybe my technic was faulty, as I gave the iodid from one to three hours before insufflating the calomel.

Hirschberg used this method also with success in conjunctival tuberculosis.

Lactic acid.—This is only to be used after curettage.

It is very efficacious on the double condition of being employed in a very concentrated solution, 75 or 80 per cent, and of being used not by mopping but upon tampons left for 25 or 30 minutes. As long as I tried mopping I only had failures.

As soon as I began using the pack, results came with unexpected rapidity.

In the course of my research I have found that this method had already been employed by Hinsberg (*Arch. of Otology*).

SURGICAL TREATMENT.

This comprises ablation of the lesions with the bone adjacent and curettage of the degenerated mucosa.

Ablation of a portion of the septum has been done by Onodi in one case of primary tuberculosis (Onodi, *Arch. int. de Laryngologie*, November and December, 1906).

This operation is quite logical. Given the great frequency of septal perforation in lupus, is it not preferable to make a perforation right at the start, going beyond the disease limits and thus giving every possible chance of cure? Esthetic exigencies only permit doing this when the lesion is such that the subsequent perforation will not be visible externally.

Some authors (Tretrop, Delsaux, Belgian Laryng. Soc., 1906) have preceded the curettage by rhinotomy, stripping up the ala nasi (Delsaux) or by median incision (Tretrop).

In Delsaux's case the operation wound was infected, for lupic nodules appeared in the scar some months after the operation.

Leaving this possibility aside, I do not believe that a rhinotomy is really advantageous for a curettement of the nasal fossae, which can be done very satisfactorily through the natural way.

Is it necessary to chloroform the patient? I think that chloroform has two clear indications: considerable extent of the lesion and fearfulness of the patient. It is plainly indicated in cases of vegetant lupus involving both the middle and lower turbinates and the corresponding part of the septum. In this manner at a single sitting all the mucosal lesions, can be destroyed, leaving only remnants to be done away with subsequently by lactic acid dressings.

Likewise too sensitive patients are spared the torment of repeating curettages, which are always painful in spite of cocaineization.

Summing up, the technic which I have adopted is:

Very limited lupus; cauterization.

Limited but vegetating lupus; curettage and lactic acid dressings.

Very extensive and vegetant lupus; curettage under chloroform.

The curettage should be painstaking to avoid recurrences. The granulations must be sought for in every place they may be, especially the edge of the vestibule, the angle between the edge and the ala nasi, and in the cul-de-sac of the vestibule, where they are easily hidden.

I apply the lactic acid dressings methodically by tamponing the nasal fossae. Between the inferior turbinate and the septum, whether these organs are involved simultaneously or separately, I introduce a wad of cotton dipped in a solution of lactic acid, 75 per cent. Two wads may be put in side by side if the space between the turbinate and septum is wide, so that the dressing will be in close contact with the lesion. A tampon may be placed in the inferior meatus if it is involved, and another in the middle meatus, above the others, if the middle turbinate is affected, always in such a manner that the dressing is in close contact with the turbinate, without compression however. The dressing is taken out after 15 to 30 minutes according to the endurance of the patient, and is repeated three times a week. The treatment is painful but bearable as a rule.

The results seem to me to have been remarkable, while they were nil with simple lactic acid moppings. The moment I began these dressings I had an improvement in Cases I, XI, XII, XIII and IX.

The mammillated infiltration disappeared very rapidly, leaving in its place a regular mucosa, smooth and of normal consistence.

The delicate point is to know exactly at what moment to stop the dressings.

It may be said that when there is doubt, the mucosa is still diseased. Every mammilla should be removed. The curette often gives valuable information by giving the soft sensation, characteristic and revelatory of lupus. In spite of everything the treatment is long and delicate. It requires much confidence and docility on the part of the patient, and much persuasion and tenacity on the part of the physician.

CASE I.

Pituitary Lupus. Lupus of Nose.

Mme. V., 29, came to Dr. Sebileau's clinic because of a constant discharge of crusts from both nares. She had no pain. But she feared that she had some serious trouble in the nasal fossae because of an extensive lupus of the nose now cured and dating ten years back. The entire external surface of the nose, almost to the root, was the seat of a thin bluish-white scar, traversed by fine capillary varicosities. The scar had been left by an extensive lupus which had produced one ulceration near the nasal bones proper, and two others about each naris which were deeply hollowed out.

This is her account of the appearance of the lupus:

After being exposed to severe cold (five hours in an open carriage, her body exposed to a biting December wind), she awoke in the morning with a swelling of the entire nose, extending to both cheeks. Some days afterward the swelling became localized to the nose, principally the nares, and became reddened. After a year ulcerations appeared. After being treated by scarification and cautery punctures without result, she went at the end of six years to Dr. Almagran, of Orleans, who cured her with sixteen x-ray treatments.

Now, five years before the nasal lupus, she noticed that the nasal secretion had become more abundant than normal. It also became dirty and slightly purulent, but had no odor.

When I first examined the patient, Sept. 1, 1902, I found: Small mammillae on the anterior portions of both turbinates, the size of large pin-heads, separated by minute ulcerated grooves. The mucosa about had lost its polish and was pale.

On the septum, anteriorly, was a mass of soft granulations the size of a pea, in the middle of which the probe penetrated the septum.

The patient's mother died of lung trouble at 24, the patient then being 4, two maternal aunts also died of tuberculosis. She had no lung lesion. Small submaxillary adenopathy, which had been much larger.

Removal of vegetations with curette and mopping with 20 per cent solution of lactic acid. Cauterization of the ends of both inferior turbinates.

In March, 1903, after several cauterizations, she seemed

completely cured. However, in July, 1903, there was a small recurrence on the turbinal ends. Actual cautery.

In March, 1904, two cutaneous lupus nodules came upon the external aspect and free border of the right ala.

The pituitary lesions reappeared at the circumference of the septal perforation and the middle portion of the inferior turbinal border. For several months I watched the patient but one day she ceased to come.

I did not see her until June, 1905. The perforation was the size of a franc piece. It had fungosities, especially on the posterior border. Both inferior turbinates had lesions, but their ends were healed. The little betraying mamillae were about the middle portions.

For eight months actual cauterizations were made at periods varying from three weeks to two months.

December, 1905, for the first time, the left middle turbinate showed a mamillated aspect which left no doubt of its involvement. The mamillae were, however, flatter than those upon the inferior turbinate, and the grooves a little larger.

Not daring to cauterize the middle turbinate, I curetted (which showed lesions of characteristic softness) and mopped with 25 per cent lactic acid. Same operation upon the inferior turbinates and the perforation.

June 9, 1906, the periphery of the perforation and the turbinates appeared to be in good condition.

November 10, 1906. New fungosities on the upper and anterior edges of the perforation. The perforation was now the size of a franc piece. The posterior border was three times as thick as the anterior and had not gone beyond the limits of the cartilage. There was evidently a tubercular chondritis. The inferior turbinates seemed cured; they contracted a little under adrenalin. The cavum, tonsils and larynx were normal. Ears normal.

Seen again January, 1907. Mamillae have reappeared on the posterior margin of the septal perforation. Application of a cotton tampon wet with 75 per cent lactic acid for 20 minutes, repeated every other day.

February 6, 1907.—Perforation is cicatrized, anterior border thin, posterior thick. Turbinates smooth and seem completely cured.

CASE II.

Pituitary Lupus. Consecutive Nasal Lupus. Bilateral Epiphora.

Mme. P., 43, came to consult me at my Amsterdam St. Polyclinic, having been sent by an associate who had treated her for a nasal lupus involving the lower two-thirds of the nose. I first saw her Nov. 23, 1903.

The lupus had begun a year previously, in October, with two or three nodules on the lobule. For four or five years she had been annoyed by a nasal trouble which manifested itself by a discharge of crusts, sometimes bloody.

She had a bilateral epiphora which came at the same time as the nasal lupus.

Rhinoscopy.—Mammillated and grooved infiltration but not in as high relief as in the preceding case. The lesion has its maximum upon the antero-superior wall of the vestibule, on each side in the angle of the ala with the septum. In this region there are also a few soft fungosities which extend slightly to the adjacent part of the cartilaginous septum. The lesions are also present, on the head of the turbinates; on the right the latter is in contact with a deviation of the septum; on the left the lupus head of the turbinate has formed a synechia with the septum very near the floor; all the anterior part of lower turbinal border is adherent to the floor, so that the inferior meatus is transformed into an almost closed sac. Lungs healthy.

Epiphora on both sides but most pronounced on the left.

The lesions were treated exclusively by galvano-cauterization, repeated every eight days. December 23 there was marked improvement in the pituitary lesions, without any change in the lacrymation.

December 30.—The turbinates look much better; but the cartilaginous septum back of the vestibular angle is involved to the extent of a 20 centime piece.

January 10 and 20.—Mopping with lactic acid. Unfortunately she was obliged to return to her home, and I ceased to see her.

CASE III.

Pituitary Lupus Beginning in Tumor Form. Consecutive Nasal Lupus. Lacrymation.

Mme. M., 59, seen in Dr. Sebileau's clinic April 24, 1904. Eight years before, she noticed interference with nasal respiration accompanied by oozing at night. At this time a physician saw a tumor in the left nasal fossa which he removed. He afterward saw mammillations of the mucosa and thought it syphilis, then gave Gilbert's syrup.

At present she has the following cutaneous lesions: Ulceration of the left surface of the nasal lobule and adhesion of the left ala to the anterior part of the subseptum; lupus nodules scattered over the nose and dorsum, especially on the left, where they reach the nasal bones proper. It was only four years ago that the lobule of the nose began to "bloat," and redden. Three years ago the ulceration and lupus nodules appeared.

The epiphora of the left lacrymal canal appeared shortly after the nasal insufficiency.

Anterior Rhinoscopy.—Both sides of the nasal septum have a mammillated infiltration of a pale blue color. The lower part of the left side of the septum and the adjacent part of the floor are especially involved. The septum has a pale bluish infiltration in this region for its entire length, almost to the posterior portion, very prominent above the healthy mucosa and composed of a lot of minute granules separated by whitish grooves. Some of these granules are united in places making little vegetations which are flabby to the probe.

The middle portion of the infiltration is ulcerated, with scooped out borders.

The extreme anterior portion of the quadrangular cartilage has a very small perforation.

The right inferior turbinate is healthy as well as the middle turbinate, same side.

The left inferior turbinate is hard to see because of the adhesion of the ala to the lobule. It seems to have the mammillated infiltration

Lungs normal to auscultation. No tubercular antecedents.

Curettage of the infiltration and septal ulcer, which were very soft. The curettage brought out actual fungosities. The

left inferior turbinate was also curetted and was equally soft. The curettage was followed by application of 20 per cent lactic acid.

Seen next, May 28, 1904. At St. Louis she had had the lobule of the nose curetted. The affection seems to have taken on a rapid course. The perforation is large and surrounded by a collarette of small friable fungosities. The right anterior portion of the septum has abundant granulations. The internal surface of the ala is much infiltrated.

But the lupus nodules of the nasal dorsum had disappeared. They reappeared later when the pituitary membrane got worse, to disappear finally and permanently with an improvement in the pituitary condition.

Curettage followed by application of 20 per cent lactic acid.

June 10 and June 23.—Another curettage of the septum and floor.

July 8.—The lupus has attacked the upper lip. The pituitary seems in good shape, except in the left vestibule. Curettage of the vestibule and application of lactic acid. Actual cautery to the lip. The patient was seen every two weeks for three months, when she was lost sight of.

February 25, 1905.—Left nasal fossa in good condition. Upper lip cured. But all the nasal lobule, or rather all the lower third of the nose, has a tubercular lymphangitis and the corresponding surface of the vestibule is vegetating. The posterior third of the left septal surface is pale as if macerated, and has a series of punctiform depressions. It is smooth and hard to the probe, and is not infiltrated. In spite of its evidently bacillary appearance and the coincidence of lupic lesions I gave specific treatment for fifteen days, without improvement.

Soon the lupus nodules appeared on the subseptum, and about the last of May it broke, weakening the lobule.

Superficial cauterizations with point on the subseptum and adjacent lip area during June and July.

September 13, 1905.—Subseptum much better. The lobule has hardly any lymphangitis. The lip is healed.

But, beside some fungosities on the lower border of the septum anteriorly, as well as the anterior border of the perforation, there was a mammillated infiltration of the left middle turbinate.

Curettage and application of lactic acid, 20 per cent.

February 14, 1906.—The lobule is weakened more and more, the subseptum being completely destroyed.

The patient was seen again March 2, 1907. The external appearance of the nose is still worse. The lobule is slightly adherent to the nasal spine. The left ala is inserted under the lobule to which it is also slightly attached. The skin of the lobule is clear of any lesion. But the vestibule is fungous. The inferior portion of the septum on the right is infiltrated back to the junction of the middle and posterior thirds. The inferior turbinate of this side is healthy. On the left there is a reappearance of lesions on the floor and middle turbinate. The left lower turbinate is atrophied.

New curettage as complete as possible. This time after the curettage a wad of cotton wet with 75 per cent lactic acid was put in and left for a quarter of an hour (the patient could not stand it any longer), and was repeated three times weekly.

CASE IV.

Primary Lupus of the Nasal Mucosa Without Coincident Skin Lesion.

Eloise B., 20, at the laryngologic clinic of Lariboisiere Hospital, March 20, 1904. Father and mother dead. Father died of bronchitis; drinker and high liver. A sister died at 20 and another at 17 of pulmonary trouble; two other sisters died at 6 months and 2 years of smallpox. She had always lived apart from her sisters.

Aside from an angina at the age of 10 she had never had any illness. She complained of difficult nasal respiration since her first year.

Rhinoscopy.—All the left vestibule is infiltrated and has soft vegetations. The septum is infiltrated with scattered vegetations the size of a lentil to a pea, finely lobulated. There is a loss of tissue extending well back. No denudation of bone perceptible to the probe. The inferior turbinate of the same side is involved, but, aside from the usual pallor of such lesions, it shows only small ulcerated grooves separating non-projecting fine lobules. The floor has the same lesions as the turbinate. No perforation of septum. Right nasal fossa clear. No odor, no headache, no pain upon pressure over the nasal bones, no spontaneous pain. No stigma of hereditary syphilis.

Four left submaxillary ganglia the size of a pea to a nut. Similar right submaxillary adenitis.

Lungs normal.

Inoculation of a guinea pig with a bit of the lesion—positive.

Lesions curetted, followed by mopping with 50 per cent lactic acid, then a series of galvanic cauterizations.

April 20, considerable improvement. Unfortunately the patient quit coming.

CASE V.

Pituitary Lupus; Consecutive Nasal Lupus.

Maria E, 16, came to the laryngologic clinic at the Lariboisiere, where she had been sent to me by Dr. Balzer, April 1, 1904. She had a nasal lupus which had been treated at St. Louis and which had originated six months before.

But pituitary lesions considerably antedated the cutaneous lesions, since for at least three or four years, she said that she began to have trouble in the right naris, blowing out crusts or picking them out with her fingers. Two years ago the same symptoms appeared in the left naris.

Anterior Rhinoscopy.—Pituitary lupus, characterized by mammillated infiltration, not much elevated above the healthy mucosa, of pale red color and lacking luster. The infiltration is especially vestibular, but it also exists on the anterior fourth of the septum and inferior turbinates, with predominance on the right. The subseptum near the lobule is likewise involved.

Numerous submaxillary ganglia. The apex of the left lung shows perhaps a slightly harsh vesicular murmur.

I omit the past history which has no essential importance here.

April 8, 15 and 22.—I made a curettage of the lesions followed by the application. At the last visit a nest of nodules in the vestibule was discovered below the tip of the lobule, with a tendency to ulceration of the septum.

May 11.—Septum cicatrized; a few suspected nodules on the right inferior turbinate and the left floor near entrance. Curettage and mopping with lactic acid.

June 14.—Another curettage of the right inferior turbinate.

June 16.—Septum completely cicatrized. The floor on both sides had suspected points which were curetted then mopped

with pure lactic acid. The general condition is also better. The patient is plainly gaining weight.

The submaxillary adenitis has almost disappeared. The patient, who does not live in Paris, was obliged to return home.

CASE VI.

*Primary Vegetating Lupus. Secondary Lupus of the Cheek Bone Following Curettage of the Mucosa.
Left Lacrymation.*

Mme. B., 42, came to the clinic at Lariboisiere November 10, 1905, for pronounced nasal obstruction.

Since the age of 10 she has been subject to coryzas lasting two or three months accompanied by a discharge of bloody crusts and disappearing in good weather. Two years ago she had been bothered by cacosmia which led her to consult a specialist, who cauterized with silver nitrate followed by application of tincture of iodine and the cacosmia disappeared in two months. He also curetted one of the nasal fossae.

A year ago the nasal fossa was curetted at Saint Antoine.

Four children living, well. Two children dead, one at 18 months of meningitis, the other of capillary bronchitis following measles. No miscarriages; no stigmata of syphilis. The lungs are sound. She has an epiphora of the left eye as a result of an abscessed lacrymal sac sixteen years ago. Did the lupus date back this far? Right and left submaxillary adenitis, voluminous in size, especially on the right, which she claimed to have had since infancy.

Rhinoscopy.—On the right the inferior turbinate is transformed into a pale vegetating mass, in contact with the septum. The mass gives the impression of something finely stippled. The vegetations are composed of a multitude of fine granulations, and are separated by depressions or even losses of tissue. The ensemble recalls the raised school geography charts. The lesion seems to go very far back, but it is impossible to say definitely because the turbinate is in contact with the deviated septum. Same lesions on floor and internal surfaces of the ala nasi on this side. On the left, same lesions.

The cartilaginous septum has a mammillated and grooved infiltration, pale, bluish, and soft to the probe. Perforation far anterior. The middle turbinates are hard to see. After

cocainization curettage of the right nasal fossa, then application of 30 per cent lactic acid.

November 16.—Curettage of left nasal fossa. When she came again on the 22nd, four days after the curettage, she had a lymphangitis over the left cheek bone with two lupus nodules. This lesion has since progressed like a lupus of the cheek.

There is no doubt in my mind that my curettage was the cause of the cheek lupus. So for a time afterward I used cauterization exclusively. But by this method the treatment took too long and I was obliged to go back to curettage, in spite of its dangers.

April 20.—Careful curettage of the right inferior turbinate and septum of same side, followed by application of 30 per cent lactic acid.

May 10.—Curettage of right inferior turbinate and septum. Beginning May 25 I cauterized the nasal fossae alternately every week until the end of June.

I saw the patient again July 20. Much improvement; the right inferior turbinate is almost healed. The left inferior turbinate is less swollen. But coming off from the left middle turbinate is seen a pale granular intumescence, soft to the probe. Both nasal fossae are completely permeable, a result which pleases the patient because they were entirely obstructed before treatment. Some fungosities around the septal perforation. Cautery stripes to the turbinates and points to the septal fungosities.

Seen again November 16, 1906. The anterior portions of both inferior turbinates are still diseased, as also the periphery of the perforation. Curettage and mopping with 50 per cent lactic acid.

January 9, 1907.—The posterior part of the left middle turbinate is enormously hypertrophied. There is a tumor hanging from it like a big raspberry, of a pale blue tint, lobulated and descending almost to the floor. The lupus of the cheek continues its course and is being treated at St. Louis.

Ablation of the tumor. Beginning at this time 75 per cent lactic acid packs were commenced. These were allowed to remain 25 minutes, and were repeated every eight days. Unfortunately after two treatments the patient quit coming to the clinic.

Result.—Patient much improved but not cured.

CASE VII.

*Pituitary Lupus. Consecutive Lupus Over Left Cheek Bone
and of the Cheek near the Horizontal Branch of the
Left Inferior Maxilla. Bilateral Lacrymation.*

Marie P., 26, came to the clinic November 4, 1905, sent to me by Dr. Balzer, in whose clinic she had been treated two years for lupus over the left cheek bone and left horizontal branch of the inferior maxilla. There were also healed lupus lesions of the lobule. The beginning of the affection was six years before. At that time she blew out crusts and had "growths of flesh" in the vestibule. Six months afterwards came the tearing of the right eye and only then the nasal lupus. Three children, well. Father and mother well. Lungs sound.

Rhinoscopy.—At present the left ala is mostly eaten out but has no lupus nodules. Left, mammillated, pale infiltration of the inferior turbinate. A few millimeters from the anterior end it has a synechia with the floor. (This is the side on which the epiphora is most pronounced.) The whole turbinate is infiltrated and has losses of substance in places. Right, the inferior turbinate has the same mammillated lesion especially along the inferior border, which is covered with purulent crusts. On both sides the inferior turbinates are greatly atrophied and the mammillae and grooves have the rifled appearance before mentioned. On both sides the middle turbinates are hypertrophied and have a very clear mammillated and grooved appearance. There is a small hypertrophied ganglion at the end of the greater cornu of the hyoid bone. left side. The treatment consisted of curettage and application of lactic acid, alternating with galvano-cauterization.

She quit February 22, 1906, very much improved, but not cured.

CASE VIII.

*Pituitary Lupus. Consecutive Nasal Lupus. Suppurative
Cervical Adenitis. Suppurative Submental Adenitis with
Lupus Transformation of the Adjacent Skin.
Lymphangitis of Nose. Scrofulous Habitus.*

Mariette R., 19, came to the clinic at Lariboisiere January 6, 1906. Mother died of hepatic cancer with ascites. Father living and well. She has cervical cicatrices, right and left,

from former suppurative adenopathies, especially behind the suprahyoid region. Occupying the entire submental region is a cutaneous lupus, developed upon a suppurative adenitis which opened spontaneously.

Three years ago she had some embarrassment of nasal respiration. She was seen at this time at Lariboisiere where a diagnosis of septal polypus was made; it was removed and the point of implantation cauterized. It was evidently a lupus tumor, as proved by the course of the case. Six months afterward there appeared two lupus nodules on the lobule, diagnosed and treated by Dr. Jonselme, who cauterized them and brought about a cure. It was about this time that the submental adenitis developed lupus. At no time did she have nasal pain.

At present the nasal lobule is somewhat puffy, and slightly red at the tip. There is a small nodule on the vestibular border of the lobule on the left side.

Rhinoscöpy.—Characteristic mammillated infiltration, non-vegetant. Right: The lesion occupies fully the anterior third of the inferior turbinate, the corresponding part of the septum, especially its vestibular portion where there is a tendency to perforate, and upon the point of the septum near the lobule. Left: Lesions less advanced; they hardly comprise the anterior third of the turbinate. The septum is involved only at its anterior portion by lesions from the lobule. On both sides the floor adjacent to the turbinates is diseased. No premaseteric adenopathy. Cavum, pharynx and larynx normal. Lungs sound. General condition very good; florid appearance. Not daring to curette for fear of making inoculations of skin lupus, I used the cautery every week in each nasal fossa, making streaks parallel to each other and crossed by other parallel streaks perpendicular to the first. February 16, 1906, the pituitary lesions are improving. But there is a lupus nodule on the posterior pillar of the velum near the upper pole of the tonsil, which I cauterized. Patient lost to sight.

CASE IX.

*Pituitary Lupus. Lupus of Nose. Lupus of Upper Lip,
Right and Left Cheek Bones. Right Epiphora.*

Gabrielle B., 38, came to Lariboisiere February 24, 1906. She had a characteristic lupus of the nose. This had begun

two years ago with two or three nodules on the upper lip at the entrance of the right naris, which spread to the right ala, then the lobule and finally almost the entire nose. Ten years ago there was epiphora of the right eye and at the same time some interference with respiration on this side. I do not hesitate to regard this as the origin of the present pituitary lesions.

Rhinoscopy.—Pronounced mammillated and grooved infiltration of the right cartilaginous septum as well as the head of the right inferior turbinate and of the vestibule at the junction of septum and left ala. In the septal infiltration is a loss of tissue the size of a fifty centime piece, with irregular borders and granular bottom. No denuded bone. No lesion in left fossa. Two weeks ago six nodules appeared over the right cheek bone. Cavum, larynx and lungs normal.

CASE X.

Pituitary Lupus. Lupus of Nose.

M, boy of 14, seen at my clinic at the Amsterdam St. Polyclinic, October 19, 1906. He has a lupus of the nose occupying much of the dorsum. Eight years ago he had lupus over the right cheek bone, which was treated unsuccessfully by radiotherapy and then excised by Dr. Nelaton, who made skin grafts from the thigh. At that time the patient complained of nasal obstruction and had pituitary lesions, for Dr. Nelaton at the time of the lupus excision curetted the pituitary.

Rhinoscopy.—Right fossa: The head and anterior third of the right inferior turbinate is transformed into a pale fungous tissue, formed of small mammillae separated from each other by minute, ulcerous grooves, bleeding slightly on being touched with the probe and not contracting under cocaine. These lesions extend diminishingly to the posterior third of the turbinate. Same lesion on the middle portion of the cartilaginous septum. These lesions are difficult to make out because of a marked atresia in the right naris due to old vestibular lesions now cured, and perhaps to former curettages. Left: The inferior turbinate has a synechia anteriorly with a spur on the septum. There is a slight mammillation of the head. The synechia bears witness to old lesions. No bone lesion.

The velum, pillars, tonsils and posterior wall of the buccal pharynx have lupus with marked infiltration. It is impossible to see the cavum. The larynx is normal. Slight submaxillary and carotid adenitis. The patient does not cough. The lungs show no physical signs.

After cocaineization I at once curetted the right inferior turbinate and septum. The lesions had a characteristic softness especially the anterior portion of the inferior turbinate. Application of 30 per cent lactic acid. Same operation on the left. The lesions are much less extensive, both as to surface and depth. Galvano-cautery points every week to the velum, pharynx and tonsils. The lesions on the turbinate and septum were treated alternately with cauterizations and curettages followed by mopping with 50 per cent lactic acid.

Beginning December 21, 1906, after a curettage of both inferior turbinates and the septum I used dressings once a week of cotton wads soaked with lactic acid, 75 per cent, which were left in twenty-five or thirty minutes. I would have liked to use dressings much oftener, but the patient would only submit to it once a week.

December 28.—The change is striking. The mucosa of the left turbinate seems to be healed. That of the right turbinate and septum, is for the most part, smooth and even except in the extreme anterior portion. Another 75 per cent lactic acid dressing, left in twenty-five minutes.

For the first time the laryngo-pharynx, which had been immune, now has six large mammillated infiltrations on the left. The left aryteno-epiglottic fold also has three lupus nodules, distinct and red, which had not previously existed: Cautery.

January 4, 1907.—Renewed application of lactic acid to the right inferior turbinate and septum for twenty-five minutes. The left turbinate no longer has any lesion. The lesions on the dorsum, which were treated at St. Louis with Finsen rays and cautery alternately, are much better.

March 12.—There remained only a few minute mammillae on the head of the right inferior turbinate.

The velum, pharynx and larynx are much improved.

CASE XI.

Pituitary Lupus Beginning on the Subseptum. Destruction of a Large Part of the Cartilaginous Septum and Both Ala. "Death's Head" Aspect. Lupus of Both Cheeks. Bilateral Epiphora.

Marie R., 46. The lupus of the nose began on the extreme anterior portion of the lobule. But, a year before the lobule lupus began, she had bloody crusts in the vestibule near the sub-septum. After the cutaneous lupus on the lobule came the lupus of the cheeks, first the right then the left. At present the ala and lobule have completely disappeared, the cartilaginous septum shows vertically between the mutilated external nasal walls—the "death's head." The entrance to the nasal fossae is narrowed by scar tissue. No pituitary lesions now. Cavum, larynx and lungs normal.

CASE XII.

Pituitary Lupus. Consecutive Lupus of Nose. Lesions of Middle Ear.

Louise B., 21, seen December 16, 1906, for nasal trouble characterized by obstruction and discharge of bloody crusts. The lupus of the nose appeared at the age of 16, in the form of five nodules on the lobule and extended to the right ala. After having had a considerable nasal discharge since the age of 10, she states that between 13 and 15, before the skin lupus, adherent sanguinolent crusts appeared upon the margin of the naris. At present the lupus of the nose is cicatrized. The right ala is partly destroyed.

Rhinoscopy.—The left inferior turbinate is somewhat atrophied, mammillated and ragged throughout. The middle turbinate also has ulcerated grooves. The septum, over almost its entire extent, has little, pale infiltrated masses, composed of very small white points separated by ulcerous grooves. Smaller mammillae disseminated all over the septum. At the middle of the septum one of these masses comes in contact with a mammilla on the inferior turbinate.

Right: The head of the inferior turbinate seems involved, but the lesions are difficult to see because of narinal atresia and a deviation of the septum. No bone lesion. Both ears

suppurating. The left was attacked with acute otitis media, following, it seems, an effort to expel crusts by blowing the nose. A mastoiditis followed for which I operated five years ago.

Both suppurating ears show identical lesions. Destruction of all the membrana except Shrapnell's. The mucosa is red and infiltrated. Slight amount of secretion.

Cavum.—Some mammillations on the upper surface of the velum, with crusts in the cavum. The facial sinuses are quite clear. Pharynx, larynx and lungs normal. After curettage under cocain I made tri-weekly application of 75 per cent lactic acid; leaving it in half an hour. February 11, 1907, the lesions seemed cured. Turbinates and septum are smooth and free from crusts.

CASE XIII.

Pituitary Lupus, Rapidly Progressing. Consecutive Nasal and Submental Lupus.

Alphonsine L., 19, was seen by me at Lariboisiere January 3, 1907. She has a nasal lupus occupying the lobule, ala and lower two-thirds of the dorsum. It is a lupus excedens with very prominent confluent nodules. Without the speculum it can be seen that all the vestibular part of the septum has been destroyed. The lobule is attached to the inferior border of the pyriform orifice only by a thin bit of cutaneous subseptum, which is also infiltrated with lupus and ready to slough off.

The beginning dates back four years. At this time there was a small swelling in the nasal fossa near the orifice which tempted the patient often to rub it. Eighteen months ago the superficial parts were attacked, and at the same time there appeared a suppurative submental adenitis which is now of a lupus character. No submaxillary, genial or premassesteric adenitis.

Rhinoscopy.—Extensive destruction of the septum comprising all the cartilaginous part, and making a perforation the size of a two-franc piece. In front it is bounded by the cutaneous subseptum, itself lupic, and below by the floor of the nasal fossae. The inferior turbinates are considerably atrophied, to a degree suggesting atrophic rhinitis. But the turbinates which are pale, are covered for the most part with pale red mammillae the size of two pin-heads, and separated from each other by grooves, some ulcerated, some cicatricial. Anteriorly

in the floor of the left nasal fossa is an eminence as large as a nut, of a pale, bluish red and very delicately mammillated, of a framboesic aspect, soft, and sessile upon the floor. In the median submental region are two erythematous plaques with lupus nodules which succeeded a suppurative adenitis, as stated. Summing up, this is a lupus with relatively rapid course without being exactly lupus vorax. Pharynx and larynx normal. Lungs healthy.

Ear.—Diminution of hearing without suppuration or tympanic alteration. Maxillary and frontal sinuses quite translucent.

Hereditary antecedents.—None.

Personal history.—At 16 had pleurisy; at 17 eruption of furuncles on legs, in ear, on chin.

January 15.—After mopping with cocain I curetted out the large mammillated mass. Part of this mass was certainly fibrous, for, besides the soft lupus lesions there was hard tissue which resisted the curette. Dressing with cotton wads dipped in 75 per cent lactic acid left in half an hour.

January 25.—Curettage of the inferior turbinates followed by the same dressing. Three times a week the dressing was renewed and left in thirty minutes. The result was excellent and quick.

February 15, 1907.—Only a month after beginning treatment, the mucosa of the floor and both turbinates was smooth and appeared to be cured.

March 9.—The fragment of subseptum has become destroyed and the lobule is sunken.

March 15, 1907.—Cure is maintained save on the posterior border of the perforation where a few granulations have reappeared. Renewed application of lactic acid.

CASE XIV.

Infected Pituitary Lupus. Nasal Lupus.

Madeline M., 14, came to the clinic January 10, 1907. She has lupus of the lobule, of the subseptum which is destroyed, and of the alae which are also mostly destroyed. The cutaneous lupus appeared only six months ago but the lesion had commenced a year ago in the "interior of the nose" where she saw "little balls."

Rhinoscopy.—On the cartilaginous septum, the inferior turbinate anteriorly and the floor, especially the left side, there is a mammillated infiltration with a vegetating tendency.

These pituitary lesions as well as the nasal ones are manifestly infected. On the nose the nodules are turgescient and the surrounding lymphangitis is considerable and quite red. The nose is very tender to touch. Same inflammation of the pituitary lesions which are red and tender to touch. There is an abundant secretion of mucus giving the lesions a varnished appearance.

For ten days I contented myself with lavages of sterile water and applying a dermatol ointment in the nasal fossae. After the lesions had about lost their inflammatory character, I curetted them under cocain, following with dressings of 75 per cent lactic acid, left in half an hour and repeated three times weekly.

March 9, 1907.—The condition of the right nasal fossa is almost unrecognizable; the septum is smooth; the inferior turbinate and floor are likewise smooth and healed. On the left there are still a few minute lesions.

The lungs, which were auscultated several times, seem sound. Transillumination of the face cavities never showed anything abnormal.

CASE XV.

Pituitary Lupus. Nasal Lupus. Tuberculous Gummi of Cheek. Suppurative Submaxillary Adenitis Causing Lupus of the Region. Submental Adenitis. Suppurative Premasseteric Adenitis.

Louise J., 30, seen February 12, 1907. She had lupus of the nasal lobule. There was considerable reddened infiltration upon which, at the upper portion, were situated seven or eight very prominent nodules. She declares that the lupus appeared only three months previously and at the same time the nasal obstruction and crusts in the right nasal fossa. The nasal lupus began with a little nodule above the right ala, near the right border of the pyriform orifice. From thence the lupus rapidly extended to the lobule. All the right side of the nose is infiltrated but without change of color. Below, the lesion has extended to the subseptum and that part of the upper lip in contact with the right naris. Although the patient (who is not very intelligent) states a simultaneous appearance of

the cutaneous and pituitary lupus. I do not believe her for the following reasons: In the first place, the pituitary lesions are far too extensive to have lasted only three months. Further, well before the appearance of the nasal lupus, all the lymphatic chain accompanying the facial was infected, proving that lesions already existed about the anterior end of the nasal fossa. Thus it was that three years previously she had a right submaxillary adenopathy, treated surgically, and a submental adenitis which suppurred. Lastly, in April, 1906, there appeared in the region of the right premaseteric ganglion a reddened nodular swelling which suppurred, leaving in its place a scar the size of two half-franc pieces.

At present the old right submaxillary cicatrix is lupic. On a line uniting the naso-genial groove with the maxillary insertion of the masseter and slightly nearer the masseter than the groove, is an inflammatory nodule encased in the skin, the size of a small pea, jutting out and surrounded by a small inflammatory zone. This nodule, situated upon the lymphatic territory of the right facial, had appeared only three or four days before.

Rhinoscopy.—The maximum of lesions is on the right cartilaginous septum, which anteriorly has a mammillated thickening the size of a franc piece, jutting out several millimeters beyond the healthy mucosa. The mammillations, which are pin-head sized, separated by filiform grooves, group in places to form small granular bunches, separated by depressions from other bunches. The whole greatly resembles a relief geography chart, to which I have already made comparison. The lesion is a pale rose and comes in contact with an analogous and very pronounced lesion on the right inferior turbinate. For this reason it is impossible to see the middle turbinate. On the left there is a slight lesion of the inferior turbinate. No bone lesion. No lesion in cavum or larynx. The frontal and maxillary sinuses are very translucent. The patient coughs. She is subject to bronchitis. Auscultation seems to indicate a slight induration in the right apex.

February 20, 1907.—Curettage of the right surface of cartilaginous septum after cocaineization. Unfortunately the patient is very timid and my cauterization was not at all thorough. Dressing of 75 per cent lactic acid, twenty-five minutes.

March 1, 1907.—Another attempt at curettage of the right

inferior turbinate after cocaineization. But I could not do it well because of the patient's intractableness.

March 9.—The middle turbinate was found to be not intact. Another fruitless attempt at curettage. Curettage under chloroform was decided upon.

CASE XVI.

Primary Pituitary Lupus. Epiphora.

Mme. X., 52, came to the Lariboisiere clinic February 5, 1907, for nasal trouble, chiefly on the left side, and characterized by a rather abundant production of crusts. She complained of lachrymation which had lasted seven or eight years. The beginning of the nose trouble dated back five or six years. Three years ago she had had a curettage of the right nasal fossa at Lariboisiere.

At present there are bilateral lesions of the cartilaginous septum. On the left is a round ulceration a little larger than a half-franc piece, flat, resting upon an infiltration which sticks out several millimeters from the healthy mucosa. The bottom of the ulceration is pale and dull. The infiltration is mammillated. Anteriorly upon the infiltration are small pimples separated by deep ulcerous grooves which may presage a perforation. On the right the lesions are of the hypertrophic type. There is a granular infiltration the size of a nut which almost touches the inferior turbinate. No lesion of pharynx, tonsils or velum. Lungs sound. Sinus quite translucent. No cutaneous lupus. Upon palpation there is felt a small, hard, premaseteric ganglion, the size of a small pea, mobile under the finger.

Inoculation of a bit of pituitary tissue in a guinea pig. A chancre developed at the point of inoculation, with caseous inguinal adenitis.

REFERENCES.

1. Cartaz. *France Medicale*, 1887.
2. Boutard. *La tuberculose nasale*, Thèse Paris, 1889.
3. Plicque. *La tuberculose nasale*, *Ann. mal. oreille, etc.*, 1890.
4. Olympitis. *La tuberculose nasale*, Thèse Paris, 1890.
5. Koschier. *La tuberculose nasale*, *Wien. klin. Woch.*, 1895.
6. Pistre. *Les pseudo-polypes tuberculeux des fosses nasales*, Thèse Bordeaux, 1903.
7. Raulin. *Lupus primitif des fosses nasales*, Thèse Paris, 1887.
8. Hinsberg. *Affections oculaires en relation avec la tuberculose de la muqueuse nasale*, *Arch. of Otology*, 1901.

9. Massel. *Rev. hebd. laryng.*, March 11, 1905, No. 10.
10. Escat. *Legitimite de la distinction clinique du lupus*, etc., *Ann. mal. oreille*, October, 1905.
11. Demme. *Zur diag. Bedeutung die Tuberkelbacillen*, etc., *Berlin klin. Woch.*, 1883, xx, No. 15.
12. Volkmann. *Ueber den Character der fungosen Gelenkentzündungen*, *Samml. klin. Vortraege*, Leipzig, 1875-1880; *Chirurgia*, Nos. 168, 169.
13. Caboche. *A propos de la tuberculose nasale*, *Ann. mal. oreille*, January, 1906.
14. Marschik. *Wien. klin. Koch.*, 1905, xviii, 22.
15. Heinde. *Wien. klin. Woch.*, 1905, xviii, 22.
16. Caboche. *Tuberculose nasale et meat inferieur*, *Ann. mal. oreille*, September, 1906.
17. Millan. *La sclerodermie tuberculeuse*, *Soc. med. hop.*, March, 1907.
18. Francis Steward. *Tuberculosis of the Nasal Mucosa*, London Guy's Hospital Rep., 1897.
19. Willigh. *Prager Vierteljahrschrift*, p. 4.
20. Weichselbaum. *Centralblatt f. klin. Chir.*, 1882, p. 26.
21. Melzi. *Arch. internat. laryngol. ital.*, etc., July and August 1904.
22. Gelle. *Role des lesions nasales dans la pathogenie du larmoiement*, *Soc. Francaise Laryngol.*, 1903.
23. Poulard. *Adenopathies dans les affections oculaires*, Thèse Paris, 1903.
24. Menier. *Manuel des maladies des fosses nasales*, Paris.
25. Chiari. *Maladies du nez*, Breyre translation, 1905.
26. Molinie. *Med. medicale*, 1893.
27. Audry. *Journal des malad. cut. et. syph.*, 1896, p. 729.
28. Mineau et Freche. *Origine nasale du lupus de la face*, *Annales Dermatologie*, 1897.
29. Sticker. *Versamml. Deutsch. Naturforschen med. Aertze*, Düsseldorf, September, 1908.
30. Bresgen. *Deu. med. Woch.*, 1887, No. 30.
31. Lacoarret. *Rev. hebd. de laryngol.*, 1892, p. 405.
32. Labit. *Rev. hebd. de laryngol.*, 1895, p. 509.
33. Gleitsmann. *Tuberculose des sinus accessoires du nez*, *Rev. hebd. de laryngol.*, June, 1907.
34. Felix. *Participation de l'oreille moyenne dans le lupus des fosses nasales*, *Ann. mal. oreille*, March, 1903.
35. Gradenigo. *Gazetta degli Ospitali*, 1888, in *Annales mal. oreille*, 1889, p. 93.
36. Pasch. *Contribution a la clinique de la tuberculose nasale*, *Arch. f. laryngol.*, Vol. xvii, No. 3, in *Ann. mal. oreille*, November, 1906.
37. Mygind. *Le lupus de la cavite nasale*, *Arch. f. Laryngologie*, in *Ann. mal. oreille*, November, 1906, p. 493.
38. Gelle. *Adenopathies dans les affections des fosses nasales* Report to the Societe Francaise de Laryngologie, 1905.
39. Garel et Collet. *Tumeurs de la cloison nasale*, *Ann. mal. oreille*, 1893, p. 249.
40. Caboche. *L'ozene forme larvee de la tuberculose*, *Sos. Francaise de Laryng.*, May, 1907.
41. Dieulafoy. *Academie de Medecine*, 1895.
42. Babes. *Penetration des bacilles tuberculeux par la peau*, *Presse Medicale*, June 15, 1907.

43. Courmont et Lesieur. Penetration des bacilles tuberculeux par la peau, Presse Medicale, 1907, No. 51.
44. Marc Andre. Lymphatiques des fosses nasales, Thèse Paris, 1905.
45. Botey. Ann. mal. oreille, August, 1906, Case VII.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINO-
LOGIC AND LARYNGOLOGIC LITERATURE.

I.—EAR.

On Vertigo and Disturbance of Equilibrium in Non-Suppurative
Diseases of the Internal Ear.

WITTMACK, Greifswald (*Archives of Otolaryngology*, Vol. xxxvi, No. 5). The functions of the labyrinth are for the cochlea the perception of sound, and for the semicircular canal apparatus the preservation of the bodily equilibrium. To diagnose disease of the labyrinth, there should be a functional disturbance in the sound-perceiving as well as in the apparatus for preserving the equilibrium. The anomaly of pronounced disturbance of hearing with the absence of all disturbance of equilibrium in many cases of nerve deafness is explained by a case of the author's where in complete absence of symptoms on the part of the semicircular apparatus, in a few weeks the patient became completely deaf and at the acme of the disease a pathologic examination showed a degenerative neuritis localized in the cochlear branch of the auditory nerve. These isolated affections of the cochlea nerve are relatively frequent while isolated disease of the vestibular nerve is very unusual, in fact there is no reported case of disturbance of equilibrium related to the semicircular canal system without simultaneous disturbance of hearing.

A symptom always suggestive of disease of the sound-perceiving apparatus, especially of the auditory nerve, is tinnitus which must fulfill these conditions; the tone must be high, continuous and very loud.

Vertigo as an aid to the diagnosis of disease of the semicircular canal apparatus can be regarded only when in the form of typical rotatory vertigo.

The reason why the auditory nerve is peculiarly affected in general diseases is explained on anatomical grounds. The cochlear branch with its associated cochlear ganglion is imbedded in an unyielding bony capsule partly surrounded by broad lymphatic spaces. The blood vessels which nourish this structure are end arteries, hence the vulnerability of the cochlear nerve to toxic substances or to any interference with the circulation.

Campbell.

On the Surgical Exposure of the Jugular Bulb.

Voss, Berlin (*Archives of Otology*, Vol. xxxvi, No. 5). The author has observed in many specimens that the jugular fossa is not found at the floor of the tympanic cavity but near its median wall and that the bulb is not infrequently situated far backward near the mastoid. In such specimens the floor and the median wall of the tympanum as high as the promontory were made up of bone which separated the jugular fossa and the carotid canal.

After completing the mastoid operation, he exposes the sinus and removes the overlying bone downward to the lowest part of the vein. He uses biting forceps and narrow concave gouges so as to avoid injuring the facial nerve. The bulb is next exposed, and to escape the horizontal semicircular canal the bone is not removed higher than 0.5 cm.

Where there is doubt as to the bulbar contents, it can be determined by aspiration; when diseased the wall is incised and the thrombotic masses removed by forceps and curettes aided by irrigation. By removal of the lateral wall of the sinus and the posterior wall of the bulb the vessel is converted into a gutter easily accessible to treatment. *Campbell.*

On Chronic Progressive Labyrinthine Deafness.

MANASSE, Strassburg (*Archives of Otology*, Vol. xxxvi, No. 5). In an anatomic investigation of thirty-one labyrinths the author found:

1. Corti's organ, including the membrana tectoria was atrophic to a stage of complete aplasia at the cochlear duct and its immediate surroundings; dilatation or constriction of the duct by distension or collapse of Reissner's membrane, hydropic degeneration of the spiral ligament, connective-tissue new formation at the lower extremity of the stria vascularis, connective-tissue and bone formation in the perilymphatic spaces, occasional atrophy of the intervening walls; constant accumulation of pigment cells in the modiolus and the rest of the cochlea.

2. In the spiral ganglion, the ganglion cells were diminished in number and shrunken. Empty spaces or connective-tissue new formation were found in their place in Rosenthal's canal.

3. In the smaller nerve channels, the nerve fibres were atrophic with connective-tissue new formation within the bony channels (perineuritis).

4. In the auditory nerve, atrophy and chronic neuritis were present with new formation of connective tissue in the nerve.

In chronic progressive labyrinthine deafness the changes in the hearing apparatus preponderate over those in the organ of equilibrium. The disease begins presumably in the cochlear nerve.

In a series of 1,033 new cases, thirty-one showed labyrinthine deafness; of these, twenty-four presented disturbance of hearing only, while in seven there was also disturbance of equilibrium.

Campbell.

Deafness of the New-born; a Disturbance of the Sound-Conducting Apparatus of the Ear.

KOELLREUTTER, Rostock (*Archives of Otology*, Vol. xxxvi, No. 6). Twenty new-born children were examined in a sleeping or half-sleeping condition. There were used a musical toy, the cricri and the following forks of the Bezold-Edelmann series C, C² and C³, and Galton whistle C⁶.

Repeated examinations were made for several days:

1. The cricri produced a distinct reaction at all times, even a few hours after birth.

2. The tones C⁶ of the Galton whistle produced a distinct reaction.

(a) All children within the first twenty-four hours after birth.

(b) Seventy-four per cent of all the children between two and four days old.

3. The tuning forks C, C² and C³ never produced any reaction.

Campbell.

Deafness in the Course of Acute Osteomyelitis and of Septic Processes in General.

SIEBENMANN, Basel (*Archives of Otology*, Vol. xxxvi, No. 6). To four cases published by others the author adds three which he has observed during the past five years.

It generally occurs during the first two decades in life, is usually bilateral. The first signs of deafness appeared always during the bone suppuration. The progress of the loss of hearing is usually very rapid. The two patients of Bezold and Castex, who became deaf in their seventh year, later became deaf mutes.

In all seven cases the middle ear escaped. In but one was

a bacteriologic examination made and this showed the presence of a pure staphylococcic infection. In acute osteomyelitis of the long bones, this bacteriologic condition is the usual one.

Assuming that labyrinthitis and not neuritis is the cause of the osteomyelitic deafness, this is supported by the fact that both divisions of the labyrinth are involved to about the same degree. The fact that in osteomyelitic deafness occasionally marked remnants of the auditory and the static function are found, suggests the condition of the labyrinth found in meningitic deafness. Moreover, the fact that during the course of the osteomyelitic deafness no perforation from the labyrinth to the middle ear has been observed, agrees with the clinical and anatomic experiences which have been collected concerning meningeal labyrinth suppuration.

Campbell.

On the Prognosis of the Operative Opening of a Purulent Labyrinth.

FREYTAG, Breslau (*Archives of Otolology*, Vol. xxxvi, No. 6). Operation on the labyrinth brings very little additional danger to the patient. The prognosis of labyrinth suppuration is improved by operative opening of the labyrinth.

Of forty-six cases observed at the Breslau Ear Clinic since Hinsberg's publication, sixteen cases of labyrinthitis were treated by exposure of the middle ear only. In these there was a mortality of 22%. In twenty-seven cases the labyrinth was opened at operation. Of these, eight cases were complicated by fatal conditions prior to operation. Of the remaining nineteen uncomplicated cases none died.

Campbell.

A Fatal Case of Sinus Thrombosis after Chronic Purulent Otitis Complicated with Cholesteatoma, Illustrating an Unusual Variety of Infection.

KNAPP (*Archives of Otolology*, Vol. xxxvi, No. 6). A boy, aged 8, suffering from kyphosis had discharge from his right ear for three years. Severe headache and vomiting existing for several months were relieved by the extraction of polypi three weeks ago. The headache returned with great severity, with vomiting, rigors and cessation of discharge. The ear-canal is collapsed, the mastoid tender and temperature 105° F. On opening the mastoid cortex it was found that the dilated antral cavity had extended into the tympanum and contained broken-down cholesteatomatous masses. There was

caries of the attic and floor of the tympanum. The sinus was exposed from bulb to knee; its wall was of a yellow-black color and when incised its lumen contained disintegrated clot, thin serous pus and gas.

On the following day there was a slight chill in the morning with a temperature of 103° F. and some congestion of the optic disc. The internal jugular, facial and thyroid veins were ligated and the enlarged cervical glands removed. The bulb was further exposed and the clot removed with a curette. With each change of dressings considerable fetid pus appeared. Septic pneumonia developed and with gradually increasing cyanosis death supervened.

Campbell.

Otalgia Considered as an Affection of the Sensory System of the Seventh Cranial Nerve.

HUNT, New York (*Archives of Otology*, Vol. xxxvi, No. 6). The ganglion of the facial nerve, the geniculate, is situated at the entrance of the aqueduct of Fallopius. Its zoster zone is to be found on the tympanic membrane, in the auditory canal, in the concha and in an adjacent marginal area of the external ear.

Otalgia is a neuralgic affection of the ear. The pain may be constant or intermittent, it may be superficial or localized in the depths of the ear. Its relative frequency to inflammatory earache is 1.8 per cent.

Reflex otalgias occur by the central and peripheral connections of the fifth, seventh, eighth, ninth and tenth nerves.

Anatomic relations exist between the facial nerve and ganglion and the trifacial nerve and its ganglion through the medium of the great and small superficial petrosal nerves.

The observation that loud and shrill sounds are accompanied by a sensation of pain in the teeth recalls the fact that the geniculate ganglion has filaments of communication with the acoustic nerve in the auditory canal.

Notwithstanding the varied nerve intercommunications, it seems clear to the author that while the facial nerve may be by no means the sole factor, it is the preponderating one in the production of otalgia.

Campbell.

Report of Two Fatal Cases of Brain Abscess.

THOMSON, New York (*Archives of Otology*, Vol. xxxvi, No. 6). Case 1. A young man, aged 18, five years previously had been operated upon for double mastoiditis. Four months

radical operation was performed and this ear gave no further trouble. On the left side he has had repeated attacks of otorrhea and three months ago dizziness persisted for one day. Preceded by a hard chill, he had severe pain centering over the left mastoid scar. Profuse discharge appeared and the mastoid swelling and tenderness increased. A radical operation was performed and the dura and lateral sinus were uncovered without showing any pathologic appearance.

This operation was followed by an attack of erysipelas of the face and scalp, but the wound granulated nicely and he was free from pain.

One month later he was suddenly seized with severe pain in the head, most severe over the frontal region. There was photophobia. Temperature up to 103.5° F. and pulse to 120. There were no localizing symptoms. Leucocyte count 6800, polymorphonuclear percentage 68. There was no marked change for eleven days, then he became drowsy, the neck muscles were rigid. Leucocytes 15,000 and polymorphonuclear percentage 92. Lumbar puncture fluid was perfectly clear and showed no growth. On autopsy a large cerebellar abscess was found which involved the entire left lobe, a portion of the central lobe and encroached on the right lobe. Its walls were indistinct and it did not show any capsule formation.

Case 2. A man, aged 50, who complained only of deafness in the left ear which was of two weeks' duration. On examination the left auditory canal was so swollen that the mt. could not be seen. There was no tenderness whatsoever, yet the anterior and posterior canal walls were incised without result.

Two weeks later he complained of dull nocturnal pain on the left side of the head followed in another fortnight by painful swelling over the root of the zygoma. On making an incision there pus was evacuated.

Two months after coming under observation, his mastoid and zygomatic region became intensely swollen, red and tender. On operation the mastoid cells were disintegrated and filled with pus. The dura above the antrum and the sinus were covered by granulations which were left undisturbed as there were no intracranial symptoms. Next day he was somnolent and two days later aphasia developed. This was followed by paralysis of the right side of the face and right arm, scanning speech and left optic neuritis. The temporo-sphenoidal lobe was explored through the mastoid wound and about one dram

of pus evacuated. His condition grew gradually worse and coma preceded death.

On autopsy an abscess cavity about the size of an English walnut was found in the temporo-sphenoidal lobe filled with thick creamy pus.

Campbell.

A Case of Acute Labyrinthitis Due to Meningitis.

YEARSLEY, London (*Archives of Otology*, Vol. xxxvi, No. 5). A man, aged 24, who at the age of $1\frac{1}{2}$ years had a serious illness during which time he put his hands to his head frequently and was continually shaking it, so that evidently he had severe head pain. He made no attempt to speak until between the age of 3 and 4 years.

Weber's test lateralized to the left:

Loud voice R = 0

L = 9 in.

Bone conduction C Fork R = 0

Left = — 32 sec.

Upper tone limit Edelman whistle R = 0

L = 5.6

The retention of the lower tone limit is due to the fact that the middle ear is intact.

Campbell.

Intratympanic Operations Under Local Anesthesia.

MILTON J. BALLIN (*New York Medical Journal*, October 5, 1907), after referring to the failure of applications of solution of cocain to fully deaden the drum, recommends in cases requiring paracentesis where a general anesthetic is not indicated or allowed, the use of a weak solution of cocain introduced into the mucous membrane of the auditory canal in the following manner: "The external auditory canal is first thoroughly cleansed with any antiseptic solution. A small piece of cotton is then placed into the canal so as to protect the drum against the ethyl chlorid spray, which would otherwise cause too much pain. Having done this, a cleft speculum is introduced in such a manner that only the superior wall of the canal is exposed to view. With the aid of an assistant a spray of the ethyl chlorid is now allowed to play on this exposed surface, and as soon as the parts are well anesthetized the needle of the syringe is quickly introduced at the junction of the bone and the fluid slowly injected as already stated; the

writer uses for this purpose 10 to 15 minims of a 1 per cent solution of cocain to which 5 to 6 minims of adrenalin solution (1-1000) have been added. The injection must be carried out very slowly so that infiltration takes place gradually, thus avoiding unnecessary pain. Having done this, little piece of cotton is then removed from the canal. We now wait for four to five minutes, at the end of which time it will be noticed that the drum has assumed a more or less bleached appearance, and has also become so insensible to pain that paracentesis can be performed without any annoyance to the patient.

Harris.

The Radical Mastoid Operation.

FRANK ALLPORT, Chicago, Ill. (*Jour. A. M. A.*, March 30 and April 6, 1907), here gives in great detail and with many excellent illustrations, the complete technic of the radical mastoid operation as performed by him. He regards the electric engine and rotary burrs most useful additions to the armamentarium.

Richards.

Some Important Surgical Relations of the Temporal Bone.

GEORGE E. SHAMBAUGH, Chicago, Ill. (*Jour. A. M. A.*, 1907). The sigmoid sinus, the facial nerve, the lateral semicircular canal, the external carotid, and the jugular bulb, should be familiar to the operator not only as to their relation to the tympanic cavity and mastoid cells, but he should also be familiar with their variations. At the tympanic orifice of the tuba auditiva the carotid is separated from the tube by a thin shell of bone which can readily be penetrated by the curette. The force of the curette can be applied with safety only along the external or anterior wall of the tube, because along its internal wall lies the canal of the carotid.

Richards.

The Treatment of Chronic Suppuration of the Middle Ear Without Removal of the Drum Membrane and Ossicles. The Duty of Restoring Hearing by Operation in Chronic Aural Suppuration.

CHARLES J. HEATH, London (*London Lancet*, April 27, 1907). Address before the West Kent Medico-Chirurgical Society, May 3, 1907. These papers are supplementary to the original paper published in the *London Lancet*, August 11, 1906. The author reports some more cases and gives the ex-

perience of other aural surgeons tending to prove his claims that the operation for chronic suppuration as performed by him conserves the hearing whereas after the radical operation or the operation of ossiculectomy, the hearing may be and frequently is worse, as the operation is done solely to stop the discharge and not at all to save the hearing. Mr. Heath considers the antrum to be the key to the position in nearly every case of suppurative ear disease and thinks if the disease in that cavity is cured that the morbid changes in the attic and tympanum will spontaneously disappear. This operation is not a simple opening up of the antrum and a clearing of that cavity and the aditus followed by drainage, as in Schwartze's operation, which caused so many disappointments and led to the institution of the radical operation, which, on the other hand, went much too far. In the new operation not only the posterior superior part of the meatus is removed, as in some other operations, but the antrum and aditus are practically eliminated, as well as all disease associated with those cavities. The whole posterior wall of the bony meatus and even its arched floor must also be removed, and this latter proceeding is followed in no other operation, but it must be done if that full access to the tympanum which is necessary both during and after the operation is to be assured. This is essential not only for the safe and thorough removal of granulations, polypi, etc., at the operation, but especially for the dressings during the after-treatment, for without the removal of the floor it is impossible to see the whole of the membrane; moreover, a pocket would be left between the arched floor and the membrane where discharge might accumulate.

Richards.

A Restudy of the Minute Anatomy of Structures in the Cochlea with Conclusions Bearing on the Solution of the Problem of Tone Perception.

GEORGE E. SHAMBAUGH, Chicago (*The American Journal of Anatomy*, August 1, 1907). As the results of his anatomic studies the author has formed a new "resonator theory" of tone perception in which he considers the vibrating mechanism of the membrana tectoria as the seat instead of the membrana basilaris as was supposed by Helmholtz. Throughout the entire length of the cochlea the mechanism of the long rows of hair cells is admirably suited to the requirements of the "resonator theory." "These hair cells are the real end-organs.

wherein the transference of the physical sound waves to nerve impulses takes place. Each cell, or rather group of cells, when adequately stimulated, leads to the perception of a particular tone. The higher tones, presumably, are taken up by the cells located in the basal coil, the lower tones by the cells near the apex of the cochlea." It is necessary to have some vibrating body to set in motion the hair cells of the organ of Corti, and for this purpose the author thinks the rigid membrana basilaris can not be the vibrating structure which it was believed to be by Helmholtz. He traced the basilar membrana through carefully made sections near the beginning of the vestibule and found that this membrane at a considerable distance from the point where the cochlea tube begins, became so thick and rigid as to preclude any idea of its being a vibrating structure. This membrane also disappears at the point in the lower end of the basal coil where the organ of Corti is still perfectly formed and this fact points to the conclusion that the vibration of this membrane is not essential for hearing. Seeking now to ascertain how impulses are transferred to the projecting hairs the question arises, can these hairs be stimulated directly by impulses in the endolymph without the intervention of the membrana tectoria? After investigating the character and relations of this most interesting membrane the author became convinced that the tectorial membrane does not float free in the endolymph above the cells forming the organ of Corti. This membrane on account of the distortion produced by the fixing and hardening agents necessary in preparing the labyrinth for histologic study has been much distorted as is shown by the various drawings which have appeared in the published works on the labyrinth. The author is certain that this membrane does not float free in the endolymph above the organ of Corti but that this position shows the one usually described as the result of the shrinking of the structure by the fixing agents. He gives a number of drawings of different sections all drawn to the same scale, showing that the hairs of the hair cells actually project into the under surface of the tectorial membrane and are not merely brought into touch with it by accident.

The membrana tectoria when examined throughout the several coils of the cochlea shows great variations in size from one end to the other, being many hundred times larger near the apex of the cochlea than at the beginning of the basal coil

and this increase in size is gradual. The membrane possesses a lamellar structure. This lamellar structure gives the membrana tectoria somewhat the appearance of a soft feather and the lamellae vary in length with the varying size of the tectorial membrane, the shorter lamellae occurring near the beginning of the basil coil and the longest at the apex of the cochlea. They are supported and held together by an apparently homogeneous semi-fluid substance, the specific gravity of which appears to be the same as that of the endolymph in which the membrane is suspended. This is shown by the fact that in spite of its semi-fluid character it sustains its position in the endolymph though supported only by its contact with the labium vestibulare and where the 'Streifen of Hensen' is in contact with the supporting cells of the organ of Corti." These characteristics of the membrana tectoria render it suitable to respond to the most delicate impulses passing through the endolymph. The great variation in size of the membrane from one end of the cochlea to the other, together with its lamellar structure, are physical characteristics which suggest the probable basis for a series of resonators which make it possible for the membrane in one part of the cochlea to respond to impulses of a certain pitch and in another part to impulses of another pitch. In this way the small impulses produced by the high pitched tones would in some way set to vibrating the tiny tectorial membrane found in the beginning of the basal coil, while the larger impulses arising from the deeper tones would produce vibrations in the much larger tectorial membrane found in the upper coils.

The vibrations in a part of the membrana tectoria produced by a particular tone must necessarily involve a considerable area of this structure. As a result a more or less extensive group of hair cells is stimulated. The nerve impulses arising from the stimulation of the several hair cells included in this group come together in the brain center of the cortex, where the tone picture forms the final step in the perception of this particular tone. When a tone slightly higher or lower than this one is produced, the same group of hair cells is stimulated, excepting for the addition of a few more cells at one end and the loss of a few cells at the other end of the area involved. The sum total of the impulses which reach the center in the brain is, therefore, different for every tone, however near they may be in the scale. The ability to distinguish between the

several tones is, therefore, a matter of education, since anatomically we encounter no difficulty in accounting for a different tone picture for each particular tone difference.

A necessary corollary of the fact that the vibration of the membrana tectoria resulting from a particular tone spreads over a considerable area of this membrane is the fact that when two tones near each other in the scale are produced simultaneously the two areas of the membrana tectoria thrown into vibration will overlap to a greater or less extent. This overlapping of the areas stimulated by several tones was first conceived by Helmholtz in his basilar membrane theory and has been looked upon by many as one of the strongest arguments in favor of his resonator hypothesis since it offers perhaps the most plausible explanation of the most difficult of all the secondary phenomena of tone perception to be accounted for, namely the phenomenon of "beats." The objections to this ingenious conception of Helmholtz which are based on the very apparently rigid inflexible character of the membrana basilaris do not hold when the delicate film-like, semi-fluid membrana tectoria takes the role of resonator.

Again, the peculiar alterations in the function of hearing which occur as the result of pathologic changes in the cochlea, find a more ready explanation on the hypothesis that the membrana tectoria is the resonator than that the membrana basilaris fills this role.

In the first place, the occurrence of "tone islands," that is, the preservation of certain circumscribed areas of hearing in cases where there has been more or less extensive destruction of hearing, finds as ready an explanation with the membrana tectoria as resonator as when we attribute this function to the membrana basilaris.

In the second place, the explanation of the pathologic phenomenon known as "diplacousis binauralis dysharmonica," where the patient hears a tone in the affected ear of a pitch different from that heard in the normal ear, is much more readily accounted for on my hypothesis. This peculiar phenomenon is readily explained as due to some slight alteration in the delicate and easily disturbed membrana tectoria which causes it to respond over a certain area to impulses of a different pitch than when in the normal condition.

Finally, an explanation of "tinnitus aurium" or subjective noises may possibly be found in this conception of the struc-

ture and function of the membrana tectoria. In the first place, the character of tinnitus aurium is usually that of an indefinite sound like the wind in the forest or the rushing of water, sounds made up of a great complexity of tones and with no definite pitch. Clinically, these subjective sounds arise from a variety of pathologic conditions. One of the best known causes of tinnitus is pressure applied to the conducting apparatus so as to push the foot plate of the stapes into the oval window. This results in tinnitus aurium of the indefinite character described above. What actually takes place when the stapes is thus forced into the oval window is an increase in the pressure of the intralabyrinthine fluid. The result of this alteration in pressure must be a disturbance of the membrana tectoria which has apparently the same specific gravity as the endolymph when the latter is under its normal pressure. The hairs from the hair cells, as I have shown above, normally penetrate into the lower surface of the membrana tectoria. Any disturbance in this membrane, however slight, would, therefore, alter the normal relations existing between the membrane and the hair cells. It seems that such an alteration from the normal relation between membrana tectoria and the hairs of the hair cells would constitute a stimulation of these cells. When the foot plate of the stapes is pushed into the oval window there would result a slight stimulation of perhaps all the hair cells in the cochlea. The result would be exactly what we meet with clinically, a tinnitus aurium of an indefinite character like the wind in the forest or the roar of a sea-shell. When a sudden increase or decrease in the blood-pressure results in tinnitus aurium the cause is the same as when the stapes is pushed into the oval window. The explanation for the increase or decrease of the intralabyrinthine pressure is here quite evident. The tinnitus aurium arising from the administration of certain drugs is also plausibly explained in the same way as due to an alteration in the blood-supply to the labyrinth with resulting alteration in the pressure of the intralabyrinthine fluid. The tinnitus occurring in Meniere's disease, where there has been an escape of blood into the cochlea, is also similarly accounted for by this conception of the physiology of tone perception. The disturbances in the function of hearing arising from an injury produced by a shrill whistle or an explosion near the ear are also readily explained. In the first place, when a permanent disturbance in hearing is thus produced it can be readily ac-

counted for by a partial severance of the relation between the membrana tectoria and hair cells so that the hairs from a greater or smaller number of these cells project free in the endolymph and do not come in contact with the membrana tectoria and, therefore, can not receive the stimulation from impulses passing through the endolymph. On the other hand, when there results from such an injury a permanent tinnitus aurium, this is explained by a partial, not complete, severance of the membrana tectoria from the hair cells over a certain area. This alteration of the relation existing normally between the hair cells and membrana tectoria may result, as we have repeatedly pointed out, in a stimulation of these cells. This explanation appears all the more rational since the pitch of the tinnitus is often approximately that of the whistle which originally produced the injury.

"To restate briefly the process by which the phenomenon of tone perception is accomplished: The sound waves conducted from the air impinge upon the membrana tympani, producing vibrations in it. These vibrations conducted along the chain of ossicles transmit impulses to the intralabyrinthine fluid through the medium of the foot-plate of the stapes. The impulses originating in the fluid in the vestibule pass directly into the scala vestibuli and through the membrane of Reissner to the endolymph where sympathetic vibrations are imparted to the several parts of the membrana tectoria depending on the pitch of the tone. The vibrations of the membrana tectoria in turn stimulate the hairs of the hair cells which normally project into its under surface. The nerve impulses originating from all the hair cells thus stimulated by a particular tone come together in the brain center in the cortex when the tone picture forms the final step in the process of tone perception."

Richards.

II.—NOSE.

Primary Nasal Diphtheria.

GEO. STIMSON (*New York Medical Journal*, December 14, 1907). After a report of several cases, one in an infant ten days old, Stimson proceeds to describe the symptoms and course of the disease and draws the following conclusions: "Primary nasal diphtheria is a more common disease than is ordinarily supposed." It is usually a benign affection, the constitutional symptoms being not marked or even entirely absent

The primary form is very seldom malignant per se, and especially is this true if it remains confined exclusively to the nares. It displays a tendency to limit itself to the nose. When it does spread to portions of the respiratory tract lower down, the severity, that is, the toxemia and systemic disturbance, seems to increase *pari pasu* with, and as a direct result of, such extension. There is a decided tendency towards chronicity. The prognosis is, as a rule, good. Paralytic phenomena, such as loss of knee jerks, paralysis of the palate, etc., are extremely rare."

It is capable of acting as a focus of infection, and the resulting cases may be of a malignant type. The importance of early detection of this disease by bacteriologic examinations of all cases of persistent purulent rhinitis of doubtful origin, especially in children, cannot, therefore, be overestimated.

Harris.

A Nasal Sporozoon.

JONATHAN WRIGHT (*New York Medical Journal*, December 21, 1907), reports the discovery of a nasal sporozoon—*rhinosporidium Kinealy*—in a specimen of benign warty growths removed from the nose of a farmer living in Tennessee. He identifies this with what has already been described by Kinealy and Beattie: "In all cases observed the organism has occurred in granulation tissue near the vestibule of the nose, which suggests the inference that the infection was carried by the finger nail. Wright describes the sporozoa as a group of exclusively parasitic protozoa. They always possess the power of reproducing endogenous minute protoplasmic nucleated corpuscle or sporules, a part of their own bodies. These are protected by a cyst envelope resistant for a time to the external environment, and without this the sporules are not reproduced or do not continue their development. He shows the distinguishing features of this particular form of sporozoon and illustrates them by several microphotographs.

Harris.

The Questionable Influence of So-called Diathetic Condition in Diseases of Throat and Nose in Children.

CHARLES GILMORE KERLEY (*New York Medical Journal*, August 24, 1907) does not believe in the so-called diathetic influence on diseases of the nose and throat and concludes as follows:

"To my mind the three factors, the sucking-in habit, the free use of cane sugar, inducing colds, and the tendency to lymphoid tissue involvement, a condition peculiar to all children, contribute largely to the production of the adenoids."

Harris.

Intracranial Complications of Diseases of the Accessory Sinuses of the Nose.

LEWIS A. COFFIN, New York, (*N. Y. Med Rec.*, November 9, 1907). Three cases coming under the author's own observation are reported and a review of the literature of the subject given. He thinks that the increase in the mortality from accessory sinus suppuration has been more apparent than real, since at present the diagnosis of disease of these cavities is made much more frequently than formerly. Operations on these sinuses should only be done by operators who are willing to sufficiently educate and prepare themselves for the work. He quotes Prof. Killian as stating that out of 100 operations on frontal, ethmoidal, and sphenoidal sinuses he had had only three deaths.

Richards.

Glottic Spasm of Nasal Origin in Adults.

FRANK A. CONLON (*Boston Med. and Sur. Journal*, August 8, 1907). The author reports a case of his own and gives histories of fifteen other reported cases. In his own case, removal of the middle turbinate stopped the attacks which were very severe. The reflex travels along the trigeminus and toward the periphery in the vagus.

Richards.

The Indications for Resection of the Middle Turbinate.

W. E. CASSELBERRY, Chicago, Ill. (*Jour. A. M. A.*, July 6, 1907). The middle turbinate is resected for the following reasons:

"To promote drainage and counter-drainage in nasal accessory sinus suppuration and for access in diagnosis and treatment.

To promote drainage and cleansing in certain types of atrophic rhinitis (atrophic ethmo-rhinitis).

To relieve edematous turgescence and to provide access for the radical treatment of non-suppurative ethmoiditis and nasal polypus.

To suspend pressure leading to headache, neuralgia, eye symptoms and broadening of the nasal bridge, etc.

To improve nasal respiration and ventilation.

To relieve hyperesthesia and to diminish certain reflexes, e. g., sneezing, asthmatic stimuli," etc. *Richards.*

III.—LARYNX.

Acute Edematous Eversion of the Ventricle of Morgagni.

T. J. REARDON (*New York Medical Journal*, June 29, 1907). Eversion of the ventricle is an exceedingly rare condition. Edema of the ventricle may accompany edema of the epiglottis or of the aryepiglottic folds. Reardon reports a case in a singer, age 40. A few days previous to the attack, she had suffered from what was diagnosed as acute follicular tonsillitis. After singing in church she became hoarse and the following day was cyanotic and dyspneic. Her voice was a hoarse whisper. Laryngoscopic examination showed the right ventricle of Morgagni everted and so edematous as to render a view of the vocal cords impossible, the mass being as large as a pigeon's egg. Intubation was performed and gave immediate relief. Twenty-four hours later the tube was removed and the larynx was found normal. The author discusses the pathology and treatment of edema and decides in favor of intubation over other measures though advising that a tracheotomy set be kept at hand. *Harris.*

Concerning the Laryngospasm of Adults.

LUDWIG NEUFELD (*Archiv. für Laryngologie und Rhinologie*, Bd. xx, Heft, ii, 1907). Laryngospasm in adults is much rarer than in children. The etiology of this condition is still very obscure, although hysteria and neurasthenia have been given as fairly frequent causes. Organic changes in the neck and larynx particularly tumors, may bring it about.

The author reports the following case: The patient, an unusually robust man, had been subject for six weeks to severe suffocative attacks, coming on once or twice every day. The slightest desire to cough brought on laryngospasm. The patient's five children were down with whooping cough. On examination nothing abnormal was found in the throat, the

larynx appearing perfectly normal. Inhalations had a good effect and after six weeks the attacks, gradually lessened.

Another case reported by the author belongs to the class of cases described as *ictus laryngis*. The etiology of this class of cases is also very obscure. The patient, a blacksmith aged 23 years, had always been well up to a year before. At that time he slept in a room in which the stove smoked a good deal. He developed a severe laryngitis which was treated. From that time until seen by the writer he had had severe attacks of suffocation.

On examination, the vocal cords were found slightly redened but otherwise the larynx was normal. Suddenly during the examination the cords closed firmly and immediately the patient fell to the floor. Cyanosis was marked, and there was an inspiratory stridor.

The attacks lasted twenty to thirty seconds, when the cyanosis gradually disappeared, and was followed by an intense pallor, with a slow pulse and dilated pupils which did not respond to light.

Such attacks occurred as often as four times a day. They could be prevented by pressure against the larynx, but came on when he laughed, or became excited. The author believes this case belongs in the class of traumatic neuroses.

Theisen.

IV.—PHARYNX.

Late Secondary Hemorrhage, Following the Removal of Adenoids.

F. L. WACHENHEIM (*New York Medical Journal*, October 26, 1907), reports two cases of profuse hemorrhage following the adenoid operation. In each instance it occurred at an interval of five days. He condemns the use of forceps as tending to leave residua and thus cause bleeding.

Harris.

Post-Operative Treatment of Adenoid Patients.

BRYAN DE F. SHEEDY, New York (*American Journal of Surgery*, March, 1908). Children must be educated in the matter of proper nasal respiration and should be put through a course of thorough gymnastics lasting over a period of one to five years. Deep nasal breathing with the mouth closed, for a few minutes at a time, eight to ten times a day, will do

much to develop air space in patients who have been mouth breathers. Expiration as well as inspiration must be done through the nose. The lips must be kept closed during sleep by strips of adhesive plaster placed over the mouth at bed time.

Richards.

V.—MISCELLANEOUS.

Ligation of the External Carotid in Rhinology and Pharyngology.

CHEVALIER JACKSON (*New York Medical Journal*, August 17, 1907) has ligated the external carotid artery thirty-eight times in thirty cases, both arteries being tied in eight cases; to arrest hemorrhage after tonsillectomy, seven cases; to forestall hemorrhage in extirpation of malignant disease of the tonsil and tongue, four cases; to forestall hemorrhage in extirpation of malignant disease of the maxillary antrum, three cases; to inhibit growth in malignant disease of the maxillary antrum, four cases; to forestall hemorrhage in extirpation of nasopharyngeal fibromata, eight cases; to arrest spontaneous nasal hemorrhage, two cases; to arrest postoperative nasal hemorrhage, two cases.

Harris.

The Etiology of Tuberculosis of the Upper Respiratory Tract.

G. B. WOOD (*Laryngoscope*, December, 1907) states that in a certain per cent of persons undoubtedly virulent tubercle bacilli will be found in the nasal cavity, but it is not possible that they remain there for any length of time. Knight states that up to 1901 there had been 108 cases of tuberculosis of the nose reported. It is very likely that a large number of cases of tuberculosis of the nose are overlooked, but even then it is remarkable that so few cases had been reported. The author, who has had an extended experience in examining the nasal cavities of cases of pulmonary tuberculosis in the Phipps' Institute, has never seen a case of primary nasal tuberculosis. It would seem that the nasal cavities are only rarely infected with the tubercle bacillus, probably because the time necessary for the propagation of this slow-growing organism permits the cilia of the nose and the nasal secretions to remove it from the fossae; but it is also very likely that the nasal secretions themselves are directly inhibitory to its growth.

Tuberculosis of the pharynx should be considered as prac-

tically identical with tuberculosis of its lymphoid tissue. When the comparative susceptibility of the various parts of the upper air passages to tuberculosis is considered, preference for liability to infection must be given to the tonsillar tissues. The results of all the research work done in relation to general microbic invasion through the tonsils, show, that in the tonsils more than anywhere else on the body surface, the penetration of the invading germ depends not so much upon any mechanical barrier presented by the tissue as upon the relative virulence of the germ.

The author, quoting from a paper published by himself in 1904, states, that the tonsils of 136 cases of pulmonary tuberculosis were examined by seven observers, and in ninety-four, tuberculous involvement was detected. The diagnosis in these cases was made mainly by histologic examination. In very advanced cases of pulmonary tuberculosis the tonsils almost never escaped infection. In nine cases reported in 1904 by the author, in which postmortem examinations were made, the tonsils in every one showed tubercles with giant cells, and in thirty-eight cases examined during the past year, he found histologic changes typical of tuberculosis in thirty-five. In a series of 1,671 cases of hypertrophied tonsils compiled from the published records of twenty-three authors, primary tonsillar tuberculosis was diagnosed in eighty-eight or 5.2 per cent. The author found the same proportion in entirely unsuspected cases when the tonsils had been removed by operation. It would seem safe to assume that about 5 per cent of children had tuberculosis of the tonsils.

The author then discusses the method of infection in pulmonary tuberculosis, particularly the liability to systemic infection from tuberculous lesions in the tonsils. If a given part of the body is inoculated, the lesion develops locally, and is generally propagated through the lymphatics of the nearest set of lymph nodes. The bacillus is arrested in these nodes, and cannot proceed further until destruction of this barrier has been accomplished.

The tonsils may be described as differentiated lymph glands, but it is not probable that they possess the same filtering qualities as the lymph nodes.

The writer has been able by rubbing a virulent culture of bovine tubercle bacilli on the surface of the tonsils of hogs, not only to inoculate the tonsils, but succeeded in finding the

tubercle bacillus in the regionary lymph gland of the neck within five days after the inoculation. In the same series of experiments he found that histologic changes appeared as early in the cervical lymph nodes as in the tonsil. In summing up, he states that the clinical importance of a tuberculous lesion in the tonsillar tissues of the throat is about the same as a local tuberculous lesion in any other non-vital part of the body which is separated from the internal organs by an intervening chain of lymph nodes.

Theisen.

Gastroscopy.

CHEVALIER JACKSON, Pittsburg, Pa. (*Medical Record*, April 6, 1907). Appearances of the stomach shown through the gastroscope are described and eight cases reported showing the value of the gastroscope as a diagnostic agent. "Gastroscopy is not simply a feat. It has a field of usefulness that will increase as our skill and knowledge increases. Gastroscopy, in my opinion, is useful for the detection and removal of foreign bodies, the diagnosis of many pathologic conditions, as inflammation, ulceration, scars, neoplasms, dilated vessels, and at least one of these conditions, namely, benign ulcer. When the gastroscope shall have gained its deserved recognition, malignant disease of the stomach will be diagnosed in many instances sufficiently early to give the abdominal surgeon a fair chance. Better still, a positive diagnosis of precancerous conditions will be made sufficiently early to enable him to save lives that are now being lost through reluctance of the patient to submit to an exploratory celiotomy. Gastroscopy is not offered as a substitute for an exploratory operation in every instance, but only in those cases in which the patient declines to submit, or in which the surgeon or the physician considers a simple procedure almost free from risk preferable to operative exploration.

Gastroscopy is the only method known today that yields ocular information as to the condition of the gastric mucosa. Other methods give information only by inference from the disturbance of function. Thus gastroscopy becomes an auxiliary."

Richards.

The Oral Cavity as a Factor in the Development of Speech.

G. HUDSON-MAKUEN, Philadelphia, Pa. Normal nasal breathing cannot always be brought about by rendering the nostrils patent or by the removal of adenoids, but in addition

to these measures, deformity of the teeth and maxillary arches must be corrected so as to allow the natural closure of the mouth and conserve the tonicity of the facial and buccal muscles. Hare-lip and cleft palate should be relieved at once, the effect on the speech being much better the earlier it is done.

Richards.

